

Effect on smoking quit rate of telling patients their lung age.

A randomised controlled trial

Gary Parkes

UCL

'I, Gary Parkes, confirm that the work presented in this thesis is my own.
Where information has been derived from other sources, I confirm that this
has been indicated in the thesis.'

.....

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Abstract

Background

Cigarette smoking is responsible for increased morbidity and mortality, but smoking rates remain high. There is a medical and political imperative to find new ways to reduce smoking rates. Previous prevalence studies have revealed a large number of smokers who have lung damage. Lung function gradually declines with age but smoking can effect the lungs as if they are aging more quickly. The concept of lung age has been known for many years but has never been investigated as a potential motivator for behaviour change.

The literature review covers the impact of smoking on chronic respiratory health, the measurement of lung function (including lung age), background information about current NHS smoking cessation therapies and available evidence for promoting behaviour change in the context of a range of psychological theories.

The research study

This thesis presents the results of a randomised controlled trial (RCT) of confronting smokers with their lung age as a complex intervention aimed at motivating people to quit. The RCT was conducted in the primary care setting. Over 500 smokers aged over 35 years were randomised to control and intervention groups. All had spirometry and those in the intervention groups received their results as lung age.

Results

At twelve months after the intervention, the smokers in the lung age group were twice as likely as the control group to have quit smoking, irrespective of the severity of their lung aging. Quit rate in intervention and control groups, respectively, were 13.6% and 6.4% (difference 7.2%, $P=0.005$, 95% confidence interval 2.2% to 12.1%; number needed to treat 14).

Conclusion

Telling smokers their lung age significantly improves the likelihood of them quitting smoking, but the mechanism by which this intervention achieves its effect is unclear. The discussion and reflective section consider the potential and actual impact of the research findings for policy, practice and future primary care research.

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1 Scope of this thesis

The core of this thesis is the planning, conduct and results of a randomised controlled trial (RCT) to investigate the effect of a complex intervention conducted in the real world of primary health care, for the benefit of smokers.

The scope of this thesis is to place this RCT in the broad context of the national and worldwide consequences of smoking cigarettes and in particular the impact on the respiratory health of individuals. Furthermore, the complex intervention presented in this thesis is placed against a background of current knowledge about what works in smoking cessation strategies. As the research is conducted in primary care in England some detailed background is given regarding Government policy and action over the past ten years to demonstrate 'where we are now'. I discuss the ways that the results of this research may be used to improve smoking cessation rates by changes to strategy, policy and clinical practice.

The intervention tested in this thesis was an existing technology which GPs were being encouraged to use, and hence the design was pragmatic rather than theory driven. However, in my literature review I have reviewed a limited number of popular behaviour theories with the purpose of giving a 'theory context' for smoking behaviour research. I have attempted to demonstrate some of the benefits, barriers and shortcomings of these theories in relation to smoking behaviour and cessation.

All research should lead to further questions and this thesis is no exception. Therefore, I also present ideas for further research and how this study could have been done differently.

This research was conducted in the busy, untidy world of general practice in suburban England and therefore I include some personal reflections on the process and practical aspects of conducting this research.

2 The policy context

2.1 Worldwide problem

According to estimates by the World Health Organisation, tobacco is the substance causing the most damage to health worldwide.¹ One third of the world population over the age of 15 years smoke tobacco, which equates to over 1000 million people.¹

Smoking seriously reduces life expectancy. In Europe smoking is the second most common cause of premature mortality (the commonest cause is high blood pressure and the third is alcohol). In the year 2000 smoking accounted for 12.3% of total years of life lost due to premature mortality.²

Smoking is the main cause of respiratory cancers including trachea, bronchus and lung. Standardised death rates from respiratory cancers are still climbing in women.² As well as being the prime cause of cancer and heart disease, it also causes many other fatal conditions and chronic illnesses among adults (see Table 1, p. 15).³

Reducing smoking-related morbidity and mortality is among the most important tasks of clinicians.⁴ Approximately one in four smokers will develop some degree of (non-malignant) lung damage.⁵ Those who are susceptible will have progressive damage through-out their life, which will only slow down if they stop smoking.⁶ Even if they do not develop chronic obstructive

pulmonary disease (COPD) they may develop other ill-effects of smoking such as coronary heart disease, peripheral vascular disease, stroke or cancer.⁷

Cancer	Lung, Upper respiratory, Oesophagus, Bladder, Kidney, Stomach, Pancreas, Myeloid leukaemia.
Respiratory	Chronic obstructive pulmonary disease, Pneumonia
Circulatory	Ischaemic heart disease, Cerebro-vascular disease, Aortic aneurysm, Myocardial degeneration
Digestive	Ulcer of stomach and duodenum

Table 1 Smoking related diseases

The harm caused by cigarettes goes beyond that of the individual smoker. Exposure to smoking home or at work can have serious consequences. In the UK it is estimated that several hundred people per year die from lung cancer caused by passive smoking.⁸ Passive smoking almost certainly contributes to deaths from heart disease which itself causes more deaths than lung cancer.⁹⁻¹¹

Even in low levels passive smoking can cause illness. Asthma sufferers are more prone to attacks in smoky atmospheres. Children are more vulnerable than adults and are exposed to smoke from adult behaviour. Children of smokers get more asthma and respiratory complications of viral infections and smoking pregnant women will reduce average birth weight, increase pre-

term birth rate and increase peri-natal mortality.^{12;13}

In summary it is clear that tobacco is a major worldwide threat to the health of smokers, families of smokers and other close contacts.

2.2 Smoking- British Government policy and personal behaviour

2.2.1 Smoking statistics (Great Britain)

The harmful effects of smoking have been recognised for many years. There is an inevitable tension between personal, public and political interests. Since 1998 there has been an increased political will to reduce smoking in Great Britain. This important milestone was marked by the Government White Paper entitled 'Smoking kills' which summarised the economic and health costs to the Nation and introduced a number of plans to achieve new targets for smoking reduction.¹⁴

At that time (1998), according to the Government White Paper, in Great Britain alone:

- over 120,000 people died per year because they smoked
- half of all who continued to smoke for most of their lives died of the habit.¹⁵
- those who smoked regularly and died of a smoking-related disease lost on average 16 years from their life expectancy compared to non-smokers.¹⁶
- smoking caused 84% of deaths from lung cancer, and 83% of deaths

from chronic obstructive pulmonary disease.³

- smoking caused 46,500 deaths from cancer a year , which was a third of all cancer deaths.³
- treating illness and disease caused by smoking was estimated to cost the NHS up to £1.7 billion per year for medical consultations, prescriptions, treatment and operations.¹⁷

2.2.2 Smoking inequalities and cost

Smoking is a major identifiable factor contributing to the gap in health and life expectancy between socio-economic groups. Even though average smoking rates have fallen over the past few decades, the rates have fallen less in the poor. For example, in 1996, one in ten men in professional jobs smoked, compared with over a third of men in unskilled manual jobs.¹⁴ The most recent figures available from government census (the General Household Survey) data show that there has been some improvement in social inequalities since the start of the 1998 plan. However in 2006, the prevalence of cigarette smoking continued to be higher for people in unskilled jobs (29%) compared to average (22%).¹⁸ Moreover the children in poorer families are more likely to suffer the health and financial burden of smoking. In the United Kingdom, children in the lower socio-economic groups are nearly three times as likely to be exposed to tobacco smoke in the home compared to households with parents who are professionals.²

Such differences are reflected in the impact of smoking on health. A higher

rate of smoking among people in manual jobs is matched by much higher rates of disease such as cancer and heart disease. Between 1991 and 1993, those in unskilled manual work were five times more likely of die of lung cancer than men in professional work.¹⁹ For the same period and age group, three times as many unskilled manual workers died of coronary heart disease than professional workers.^a

In 1989, it was estimated that smoking cost the NHS approximately £1.7 billion per year.¹⁷ The cost was high in both ill health and loss of productivity. It was estimated that the country lost 50 million working days per year (about 1% of the total).

Increasing taxation on cigarettes can produce a reduction in smoking. A 10% increase in taxes leads to an estimated 4% cut in smokers or smoking. However the impact on poor households is magnified due to the disproportionate numbers of smokers on benefits or low income. Mothers with dependent children who receive income support are more than twice as likely to be smokers than average.² Remarkably, poor households may spend as much as 15% of their disposable income on smoking. Smoking prevalence can be regarded as a marker of deprivation.²⁰

2.2.3 Smoking- Government plans and targets

In 1997 the British Government expressed its determination to tackle the problem of smoking. At that time, well over a quarter of the people of Britain

^a 81 per 100,000 professional workers died from coronary heart disease compared with 235 per 100,000 in unskilled manual jobs (1991-1993).

smoked. Whilst maintaining the right of individuals to make decisions they declared that:

‘Smokers have a responsibility to themselves - to their own health, and to ensure that in making the choice to smoke, their choice is based on a real understanding of the risks involved. With their right to smoke, too, comes the responsibility to others who choose not to smoke. Just as the Government is determined not to infringe upon people's rights to make free and informed choices, it is also determined to ensure that the responsibilities of smokers to people who choose not to smoke are carried out. That means a balance of rights and responsibilities -for those who smoke and for those who do not. Striking that balance is a clear and tough challenge - for the Government, for business, for local authorities, for voluntary groups and especially for individuals.’¹⁴

Through a series of proposals for legislation and improved NHS smoking cessation programme provision they created targets:

‘To reduce adult smoking in all social classes so that the overall rate falls from 28% to 24% or less by the year 2010^b; with a fall to 26% by the year 2005. In terms of today's population, this would mean 1.5 million fewer smokers in England’.^c

^b This target was for improvements measured against a baseline of 28 per cent smoking prevalence among men and women aged 16 and over in 1996.

^c The objective is not only to see smoking in all socio-economic groups reduce to a new average figure of 24% by 2010, but also to reduce the difference in smoking rates between manual and non-manual groups.

2.2.4 Smokers –Government action

A large part of the Government action concentrated on legislation, advertising bans and presentation of unpleasant words and images on cigarette packets. The intention of anti-smoking legislation is to reduce tobacco consumption and to create less exposure and a more supportive environment for those smokers who would like to quit.

Bans on workplace smoking have been effective in reducing prevalence of smoking.²¹ Remarkably, following bans on smoking in public places the new rules have widespread public support and smoking has become socially less acceptable in some countries including Ireland and the UK.¹⁸ Four out of five people agree with the ban of smoking in public places according to the General Household Survey.¹⁸ This top-down approach with government legislation about public places and restrictions on advertising and health warnings are ways of trying to force behaviour change.^{22;23} However, early evidence is supportive of the ban of smoking in public places and has led to a 5.5% fall in the numbers smoking in the 9 months after the ban (compared to a fall of 1.6% in the 9 months before the ban) and positive support of over 60% of the population.²⁴ It is too early to determine the sustainability of this fall.

More resources have been put into helping those who have decided to quit smoking and NHS clinics to support potential quitters and increased use of medication to reduce the unpleasant physiological and psychological barriers to change. In 2007 the net ingredient cost of pharmacotherapies used to help

people stop smoking was over £60 million and a similar amount was spent on NHS Stop Smoking Services.¹⁸ This represented a sevenfold increase in the expenditure on smoking therapy over 5 years.¹⁸ While costs of helping people stop smoking have increased the estimated expenditure on NHS treatment of smoking related disease has dropped to £1.4 billion from £1.7 billion in 1989 (see p. 18).¹⁸ The implication is that the policy is working even if progress is rather slow.

2.2.5 The 'right to smoke' lobby

Some controversy and opposition has arisen to the increasing trend to regard smoking as antisocial and as a reaction to anti-smoking legislation. Some writers regard smoking as a 'public good'. Here is one point of view:

'Tax is levied on tobacco in three ways: excise duty at a specific rate per 1,000 cigarettes, an additional rate based on 20% of the total retail price, plus VAT at 17.5% of the final price - including the other taxes. The end result is that tobacco taxation, the amount levied in various ways by the government on every packet of cigarettes, cigars or smoking tobacco, comes to £12 billion per year, six times more than any NHS bills run up by nicotine addicts. In fact, between 80% and 90% of the cost of a packet of fags is tax'.²⁵

It is likely that those who continue to smoke will be increasingly ostracised to the point that there will be polarisation of opinion among the public with smoking seen as antisocial by the majority and illegal in many places, but with a hard core of dedicated smokers opposed to legislation and bans.

2.2.6 Future developments

Although prevention should be the ultimate aim (i.e. discouraging young people from starting to smoke) there remains a large cohort of smokers who have not committed themselves to quitting or who have considered quitting but not been able to succeed. Nearly 80% of smokers in the UK have an intention to quit,^{18;20} but wanting to quit is not the same as quitting.²⁶ In studies investigating predictors of quitting the most frequently cited reasons for quitting were concern for current and future health (92%) and cost (59%).²⁷ Any new strategies that can move people towards a decision to quit and then to successfully abstain from smoking will be of potential benefit.

2.3 Health service standards

2.3.1 The National Institute for Health and Clinical Excellence (NICE)

Guidelines for the diagnosis and management of chronic obstructive pulmonary disease (COPD) were produced in March 2004 by the National Institute for Health and Clinical Excellence (NICE) and published as a supplement to *Thorax*, the journal of the British Thoracic Society.²⁸ NICE has also produced guidance on brief interventions for smoking cessation.²⁹

The National Institute for Health and Clinical Excellence (NICE) is the independent organisation established by the British Government by the merger of the Health Development Agency and the National Institute of Clinical Excellence in April 2005. Their stated aim is to be responsible for providing national guidance on the promotion of good health and the prevention and treatment of ill health. NICE guidance is developed by a number of independent advisory groups made up of health professionals, those working in the NHS, patients, their carers and the public.

In general there are three main areas of guidance that are produced by NICE: public health, health technologies and clinical practice. The development of all clinical practice guidance follows a common pathway. The selection of topics can be based on recommendations by any interested party

but have to comply with various criteria including the following:

- burden of disease (population affected, morbidity, mortality)
- resource impact (i.e. the cost impact on the NHS or the public sector)
- policy importance (i.e. whether the topic falls within a government priority area)

First the Department of Health (DoH) refers the topic to NICE. Once the topic has been selected, stakeholders register their interest and the National Collaborating Centre (NCC) is commissioned to prepare the scope of the guideline. Within the scope of the guideline a group made up of health professionals, representatives of patient and carer groups and technical experts start to collect evidence and develop recommendations. There is at least one public consultation period for registered stakeholders to comment on the draft guideline. An independent guideline review panel reviews the guideline to check that stakeholder comments have been taken into account.

The scope of the guidelines for chronic obstructive pulmonary disease (COPD) includes diagnosis, drug therapy and other management and it grades the recommendations by the levels of evidence available. Evidence is classified according to the types and quality of research studies that have been published.³⁰ NICE then grades their recommendations based on this hierarchy (see Table 2, p. 26).

	Hierarchy of evidence ^d		Grading of recommendation ³⁰
1a	Evidence from systematic reviews or meta analysis of randomised controlled trials	A	Based on hierarchy 1 evidence
1b	Evidence from at least one randomised controlled trial		
IIa	Evidence from at least one controlled study without randomisation	B	Based on hierarchy II evidence or extrapolated from hierarchy I evidence
IIb	Evidence from at least one other type of quasi experimental study		
III	Evidence from non experimental descriptive studies, such as comparative studies, correlation studies and case control studies	C	Based on hierarchy III evidence or extrapolated from hierarchy 1 or II evidence
IV	Evidence from expert committee reports or opinions and /or clinical experience of respected authorities	D	Directly based on hierarchy IV evidence or extrapolated from 1,II or III evidence
DS	Evidence from diagnostic studies		Evidence from diagnostic studies
NICE	Evidence from NICE guidelines or Health Technology Appraisal programme		Evidence from NICE guidelines or Health Technology Appraisal programme

Table 2 Hierarchy of evidence and NICE

The following extracts are taken from the 2004 NICE guidance for COPD and have some relevance to the background for this randomised controlled trial.

^d This was based on the structure of the Scottish Intercollegiate Guidelines Network current in 2004 and reproduced in the NICE guidance for COPD 2004. The SIGN hierarchy of evidence was last changed in June 2008.³¹

1. 'All COPD patients still smoking, regardless of age, should be encouraged to stop, and offered help to do so, at every opportunity' (Grade A evidence).²⁸
2. 'COPD can be present in the absence of symptoms' (Level III evidence).³²
3. 'A study of opportunistic case finding, found that 27% of patients who were aged over 35 years, were current or ex-smokers and had a chronic cough had reduced FEV1' (Level III evidence).⁵
4. 'Spirometry should be performed in patients who are over 35, current or ex-smokers, with a chronic cough' (Grade D evidence. Recommendation number 20).
5. 'Spirometry is fundamental to making a diagnosis of COPD and a confident diagnosis of COPD can only be made with spirometry' (Level IV evidence).
6. 'Opportunistic case finding should be based on the presence of risk factors (age and smoking) and symptoms. The diagnosis should be confirmed using spirometry' (Level IV evidence).
7. 'COPD can be detected by opportunistic case finding in primary care' (Level III evidence).^{32 33;34}
8. 'Knowledge of abnormal lung function as part of a motivational package, significantly affects the success of smoking cessation therapy' (Level Ib evidence).^{35;36}

It is noteworthy that the levels of evidence for most of these statements and recommendations are at a level D or IV. This is compatible with lower levels

of evidence based on opinions and expert committees (see Table 2, p. 26).

Part of the problem with the NICE guidance on COPD is that it had an extremely broad remit with a limited amount of time. In a rapidly changing research environment guidelines may be out of date by the time they are produced. In contrast, guidelines based on low-level evidence highlight the fact that much of the evidence required either does not exist or are of too low quality to give robust evidence based advice (see points 4, 5, 6 and 7 above).

Next, politics cannot be altogether excluded from production of guidelines that have resource implications. NICE has a remit to include cost benefit economic analyses whereas the Scottish Intercollegiate Guidelines Network do not.³¹ NICE guidance is therefore tempered by the cost and the benefit of widespread implementation. Although it is desirable and right to encourage stakeholders to have significant input to guidance, vested interests from pharmaceutical companies, specialist charities and others may have significant effect on the flavour and affordability of the guidance due to their individual agendas.

One worrying point in the NICE guidance for COPD is the quality of analysis of the available evidence. For example the NICE document claims that the evidence that 'Knowledge of abnormal lung function as part of a motivational package, significantly affects the success of smoking cessation therapy'- is level 1b in the hierarchy, which equates to 'evidence from at least one randomised controlled trial'. Their claims are not substantiated by the limited

evidence cited and are not consistent with a Cochrane review published a year later. This will be discussed in detail in Section 3.4.2 (see p.78). Moreover whilst Cochrane reviews are usually focussed on evidence relating to a specific research question, NICE guidance is very broad in scope and perhaps this is responsible for the detriment to quality.

The NICE guidance on brief interventions for smoking cessation considers whether brief smoking cessation interventions are effective at encouraging individuals to quit smoking.²⁹ The impact of wider policy and practice on smoking cessation will be the subject of a future document by NICE. By their definition:

‘Brief interventions are health promotion by a range of primary and community care professionals. For smoking cessation, brief interventions typically take between 5 and 10 minutes. The particular package that is provided will depend on a number of factors, including the individual’s willingness to quit and how acceptable they find the intervention’.²⁹

Brief interventions include one or more of the following:

- simple opportunistic advice to stop
- an assessment of the patient’s commitment to quit
- an offer of medication or behavioural support, provision of self-help material and referral to more intensive support such as the NHS Stop Smoking Services.²⁹

They assert that help that is offered should depend on a number of factors, which include the smoker’s willingness to quit, acceptability of the different

interventions available and the previous methods used.

In conclusion within the whole thrust of the guidance from NICE is the underlying principle that the only reliable way of preventing deterioration of chronic progressive lung damage from smoking, is smoking cessation.³⁷ This concludes the main recommendations so far from NICE guidance for COPD relevant to this thesis. This and other evidence about smoking cessation will be discussed in more detail in the first literature review (see Section 3, p.36).

2.3.2 National service framework (NSF) for COPD

National service frameworks (NSFs) are a rolling programme launched by the British Government in April 1998, which cover many different clinical areas.

*NSFs set long-term strategies for improving specific areas of care. They set measurable goals within set time frames.*³⁸

In particular NSFs:

- set national standards and identify key interventions for a defined service or care group
- put in place strategies to support implementation
- establish ways to ensure progress within an agreed time scale
- are one of a range of measures to raise quality and decrease variations in service, introduced in the document: 'The New NHS and A First Class Service'. The NHS Plan re-emphasised the role of NSFs as drivers in delivering the Modernisation Agenda.

On the 28 June 2006, the Secretary of State announced that a National Service Framework (NSF) should be developed for Chronic Obstructive

Pulmonary Disease (COPD) following recommendations published in the Chief Medical Officer's Annual Report of 2004.

To ensure that the NSF meets the needs of COPD patients and their carers an External Reference Group was established to produce final advice by the winter of 2007. The NSF for COPD will be published at the end of 2008 and implementation is planned for the beginning of 2009.³⁸ The contribution of this research thesis to potential changes to policy will be discussed further in Section 8.7 under the heading of 'Government policy'.

2.3.3 Quality and outcomes framework (QOF)

The new General Medical Services (nGMS) contract is the title of the agreement between the British Government and general practitioners which was implemented in 2004. The new contract moved away from the old item-of-service method of calculating pay and proposed to pay doctors for reaching quality targets.

The following principles are among those agreed by the negotiators for inclusion of indicators in the QOF.³⁹

- Indicators should, where possible be based on best available evidence.
- The number of indicators in each clinical condition should be kept to the minimum number compatible with an accurate assessment of patient care.
- Only data which are useful in patient care should be collected. The basis of the consultation should not be distorted by an over-emphasis

on data collection.

With respect to chronic obstructive pulmonary disease the nGMS contract included some incentives to promote the wider use of reliable diagnostic measurements, follow up of patients and clinical recording of smoking habits. In particular the supporting documents for the new GMS contract 2003 contained eight indicators for COPD. They outlined the rationale for inclusion of COPD and the choice of quality indicators. Each of the indicators was given similar value and, if target levels were reached overall, accounted for a maximum of 45 points out of a total of 1000 points in the whole system. The eight indicators were as follows:

- 1. The practice can produce a register of patients with COPD.
- 2. The percentage of patients in whom diagnosis has been confirmed by spirometry including reversibility testing for newly diagnosed patients with effect for 1st April 2003 (target 90%).
- 3. The percentage of all patients with COPD in whom diagnosis has been confirmed by spirometry including reversibility testing (target 90%).
- 4. The percentage of patients with COPD in whom there is a record of smoking status in the previous 15 months (target 90%).
- 5. The percentage of patients with COPD who smoke, whose notes contain a record that smoking cessation advice or referral to a specialist service, if available, has been offered in the past 15 months (target 90%).
- 6. The percentage of patients with COPD with a record of FEV1 in the

previous 27 months (target 70%).

- 7. The percentage of patients with COPD receiving inhaled treatment in whom there is a record that inhaler technique has been checked in the preceding 2 years (target 90%).
- 8. The percentage of patients with COPD who have had influenza immunisation in the preceding 1st September to 31st March.

The criteria set for the spirometric diagnosis of COPD were different from both the British Thoracic Society and GOLD guidelines. Both of these well-established guides indicate that FEV1 should be below 80% of the predicted value for diagnosis of COPD to be made among other criteria (see Table 3, p. 41). However, the QOF required the FEV1 to be less than 70% for inclusion in the register. The rationale given for this diversion from well established practice was:

‘a significant number of patients with a FEV1 less than 80% predicted may have minimal symptoms. The use of 70% enables clinicians to concentrate on symptomatic COPD.’

The implications of this policy and the relevance of the results of this study are discussed in the section on Government policy (see Section 8.7, p. 225).

Smoking indicators were not included as a distinct category in the QOF of 2003. In particular there was no requirement for an overall smoking register or indicators of promotion of smoking cessation. Instead the QOF included targets for recording of smoking status (in the previous 15 months) and smoking cessation advice or specialist referral (being offered in the previous 15months) for people who had specific diagnosed disease categories caused

by or made worse by smoking. These two indicators were listed under the disease categories of coronary heart disease (total 11 points), stroke and transient ischaemic attack (total 5 points), and hypertension (total 20 points), diabetes mellitus (total 8 points), asthma (total 12 points) and COPD (total 12 points). Therefore a sum total of 68 (out of the 1000) points were offered as a financial incentive for reaching 90% targets of recording smoking in each disease category and either 70 or 90% target of giving smoking cessation advice or referral.

Since the initial development of the QOF indicators a number of minor changes have been made but the total number of points available (68 points) under the categories of COPD and smoking remain the same. Quality criteria 2 and 3 have been combined into a single criterion of the percentage of those with a diagnosis of COPD in whom diagnosis has been confirmed by spirometry. There has been an important change to the diagnostic criteria. Reversibility is no longer included as an indicator which had been a controversial inclusion since 2004 as it was out of line with NICE recommendations and with other international standards.³² The criteria for diagnosis were changed in the revision of the contract for 2008/09 to 'confirmation by post-bronchodilator spirometry', which is consistent with American Thoracic Society and European Respiratory Society (ATS/ERS) recommendations (see Table 3, p. 41). Moreover the FEV1 level (to below 80% of predicted) for diagnosis is now compatible with British Thoracic Society Guidelines but not with the ATS/ERS guidance.

Despite the fact that the true prevalence of COPD has been difficult to establish and estimates have been wide,⁴⁰ the Government has also factored-in a financial reward for those practices who have identified sufficient patients with COPD in line with national and regional prevalence rates. Most estimates of COPD prevalence from research data indicate that there are a large proportion of those with COPD are as yet undiagnosed.⁵ Therefore, I believe this to be a useful development for the QOF to improve the quality of the disease registers and to increase case identification.

In line with the negotiated principles, it is important that the structure and payment incentives of QOF reflect the current levels of evidence for smoking cessation strategies and prevention and treatment of COPD. The background principles that should be used for the QOF indicate that any changes should be directed by clinical evidence and therefore any new clinical data needs to be considered during reviews and in contract negotiations. It will be important to disseminate any new information and evidence from this study and others to gain maximum beneficial impact for smokers and for the best use of finite resources of the health service.

3 Literature review 1: General background on smoking cessation

3.1 Chronic obstructive airways disease and smoking

Smoking is the most common cause of chronic obstructive pulmonary disease (COPD).⁴¹ The burden of disease from COPD is enormous. Population studies in the United Kingdom indicate a prevalence of 14% among adults.⁴² According to official statistics 26,000 people died of COPD in England and Wales in 1999.⁴³ The British Thoracic Society (BTS) estimates that there are as many as 200 patients with COPD on each General Practitioner's list. Consultation and admission rates are high among those 200 patients on each GP list, and several of them will die each year. Worldwide it is difficult to estimate the prevalence of COPD. Information from collated data gives an estimated prevalence of between three and ten percent and it is the fourth most common cause of death worldwide.^{40 44}

It is now acknowledged that the most reliable way of diagnosing COPD is using the results of spirometry (see Appendix 1. Glossary, p.286) although there is still some debate about the diagnostic criteria.^{45;46} The British Thoracic Society has been calling for the widespread use of spirometry for many years but uptake has been slow until recently.⁴⁷ The technology for diagnosis is now increasingly available in primary care in the UK and many

other developed countries. Undoubtedly the changes to the General Medical Services contract have encouraged wider use of spirometry in the UK, as there are now financial incentives for confirming diagnosis and for regular monitoring of disease progression using spirometry (see Section 2.3.3, p. 31).

In the early stages of COPD there may be few if any symptoms and therefore symptom scoring strategies have been found to be unreliable for very early detection. However spirometry can detect changes in the lungs even before typical symptoms of breathlessness, cough or wheeze become apparent. In cross sectional surveys of smokers over 40 as many as a quarter may have previously undetected lung damage even in the absence of symptoms.⁴⁸

Many years of research have established that drugs and other techniques have little impact on disease progression if people continue to smoke. The most important factor in slowing down deterioration of the lung damage is to stop smoking.^{4;49} Even the most recent results of combination inhaled drug therapy (TORCH study) have been disappointing.^{50;51}

The TORCH study (TOwards a Revolution in Chronic obstructive pulmonary disease) recorded smoking related deaths in those with moderate COPD.⁵⁰ They defined moderate COPD as a forced expiratory volume less than 60% of predicted value and FEV1/FVC ratio of less than 0.70 and selected smokers with at least ten pack-year history. This was a randomised double blind trial of over 6000 patients comparing different inhalers against placebo.

The main aim of the study was to compare mortality for different combinations of inhalers. The revolution that they hoped for proved to be a misnomer as combination treatment of COPD with fluticasone and salmeterol did not reduce mortality in those with COPD compared to placebo. One in eight of those with COPD died in the three year study and nearly half of the deaths were judged to be attributable to smoking.⁵⁰ Therefore, this not only illustrates the high mortality in this group but also that drug therapy does not yet hold the key to improving mortality in those with COPD.

It is widely acknowledged that COPD is unrecognised in large numbers of smokers and ex smokers.^{52;53} Many authorities believe that obstruction can be detected a long time before the patient becomes aware of their limitations or develops typical symptoms such as cough, shortness of breath or wheeze.^{46;49} Late diagnosis and misdiagnosis of COPD is common in the UK. Surveys of asthma registers in United Kingdom general practice have revealed that many people are misdiagnosed on asthma registers.⁵⁴ Up to three quarters of people with COPD remain undiagnosed and it is common for diagnosis of damage to lung to be delayed for up to 20 years from the time of detectable changes.^{5;37;55} Lung changes can be detected after 20 pack years of smoking. The majority of people start smoking when they are in their teens and therefore likely to show changes as early as the age of 35 years. Some studies have even detected changes in college students aged under 25 years, after only a few years of smoking although these findings have not been replicated elsewhere.⁵⁶

People do not necessarily seek medical help even if they have symptoms suggestive of COPD. In one study a random sample of the public were screened. Of the people with newly detected COPD only a third of them had ever consulted a doctor about their symptoms.^{44;57} Moreover, the average age of diagnosis of COPD in the United Kingdom is 55 years.³⁷ This 20-year delay in diagnosis offers a potential large window of opportunity for earlier detection and possible intervention to reduce morbidity.

Many authors have proposed screening of smokers in the pre-symptom or early symptomatic stage so that intervention can be concentrated on those likely to benefit most.^{58;59;60} However, other authorities maintain that there is insufficient evidence of benefit with early detection and resources should be concentrated on those with established symptomatic disease.⁶¹ These issues will be discussed further in a later section on the pros and cons of screening (see Section 8.7.2, p.227).

3.1.1 Lung damage and its measurement

As explained in the previous section, although COPD is the diagnostic label of a group of conditions typically characterised by respiratory symptoms of shortness of breath, cough and sputum production in combination with airflow limitation (obstruction) and chronic inflammation of the lung, in the early stages there be minimal or no symptoms.³² Therefore it is vital to have objective diagnostic testing of those suspected of having lung damage from smoking. Spirometry is now regarded as a vital tool in the assessment of severity of obstruction in lung disease (COPD and asthma) as well as being useful in distinguishing these diseases from other causes of respiratory

symptoms.⁴⁷

The British Thoracic Society (BTS), Global Initiative for Chronic Obstructive Lung Disease (GOLD) and the American Thoracic Society/ European Thoracic Society (ATS/ERS) have each produced criteria for the diagnosis of COPD using spirometry at the centre of diagnostic testing.^{47;62} The main measures of lung function are FEV1 (forced expiratory volume in 1 second) and FVC (forced vital capacity) (see Appendix 1. Glossary, p. 286).

The current diagnostic spirometry criteria for COPD (Table 3, p.41) of these three influential organisations are listed and compared as follows:

- BTS British Thoracic Society.³²
- GOLD Global Initiative for Chronic Obstructive Lung Disease.⁶³
- ATS American Thoracic Society/ European Thoracic Society.⁶²

Table 3 Diagnostic criteria for COPD

	BTS		GOLD		ATS and ERS (Post broncho- -dilator FEV1)	
	FEV1 % predicted	FEV1/FVC	FEV1 % predicted	FEV1/FVC	FEV1 % predicted	FEV1/FVC
Mild COPD	50 to 80%	<0.7	≥80%	<0.7	≥80%	≤0.7
Moderate	30 to 49%	<0.7	≥50<80%	<0.7	50 to 80%	≤0.7
Severe	<30%	<0.7	≥30<50%	<0.7	30 to 50%	≤0.7
Very severe			<30%	<0.7	<30%	≤0.7
Very severe			<50% + respiratory failure	<0.7		
Reference values used	ECCS ⁶⁴		ECCS ⁶⁴		NHANES 3 ⁶⁵	

There is considerable dispute about the value and technique of ‘reversibility testing’.^{66;67} By definition, those with COPD have obstruction of the airways which is mostly irreversible and have very little day-to-day variation in symptoms or FEV1/PEFR measurements on spirometry. Asthma should be distinguishable from COPD by the tendency for variable wheeze and breathlessness and a significant change (15% and 400ml)^e in FEV1 after

^e If the FEV1 improves by 15% but < 400ml the subject is said to have COPD with reversibility not asthma.

using drugs to maximise broncho-dilation. Other researchers have thrown doubt on the value and use of reversibility testing.⁶⁸ However, between countries and even within the United Kingdom the techniques of reversibility testing are very varied. A survey of hospital lung function laboratories revealed the use of a wide variety of different drugs, doses and delivery systems (for the drug) used as well as wide variations in repeat spirometric testing for reversibility.⁶⁹ The BTS has attempted to standardise the technique but there has been little consistent use.⁴⁷

There is a move away from routine use of 'reversibility' as part of spirometry testing. Doubts have been thrown on the usefulness of 'reversibility' as a theoretical or practical tool. There are various reasons for this change. First there is the lack of evidence that a 15% change of FEV1 after administration of bronchodilators is predictive of a subsequent response to treatment of COPD with steroids or beta agonist and therefore it is argued that the distinction is irrelevant. Secondly, the diagnosis of asthma can be made by clinical features with assessment of variability of airways obstruction with serial peak flow measurements. Finally, in the United Kingdom the General Practitioners' contract was revised in 2008/9⁷⁰ in a move towards the ATS/ERS⁶² method of assessment, which uses post-bronchodilator measurements as the standard measurement of FEV1.⁶² However they still use different reference tables and definitions (by percentage of predicted value) for the different levels of severity of disease.

It is important for any intervention to be conducted by the most appropriately trained personnel in the most economic way consistent with good standards of care. Training of primary care assistants and the early detection of new cases of COPD by screening in primary care is possible, quick and inexpensive.^{5;71}

A single measurement of FEV1 cannot completely represent the complex clinical consequences of COPD because many patients have few or very subtle symptoms and persistent cough and sputum production may precede the development of airflow limitation and the first symptom may be the development of shortness of breath with previously tolerated activities.

The Medical Research Council dyspnoea scale can be used to assess functional dyspnoea as follows in Box 1:

0. Not troubled with breathlessness except with strenuous exercise
1. Troubled by shortness of breath when hurrying or walking up a slight hill.
2. Walks slower than people of the same age due to breathlessness or has to stop for breath when walking at own pace on the level.
3. Stops for breath after walking about 100 metres or after a few minutes on the level.
4. Too breathless to leave the house or breathless when dressing or undressing.

Box 1 MRC dyspnoea scale

Classification of COPD using all the different components of the disease (e.g.

weight loss, breathlessness, impact on life) is not yet available. Other symptom and impact scoring systems will be discussed later (see Section 5.6, p. 176).

Lung age

Normal lung function gradually reduces throughout adult life. This was documented graphically by Fletcher and Peto in their prospective epidemiological study of lung function in men, 30 years ago.⁶ They showed that forced expiratory volume in one second falls over time (red line in Figure 1) but in non-smokers and in many smokers (who are apparently not

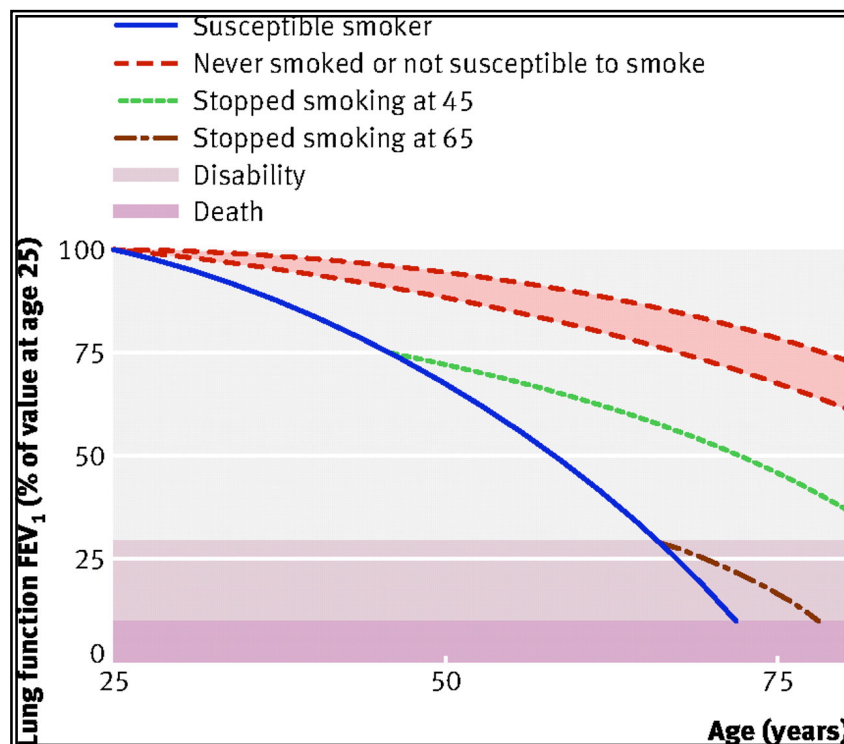


Figure 1 Lung function with time and smoking

(Adapted from Fletcher and Peto)⁶

susceptible), significant airflow obstruction never develops. However, in susceptible individuals irreversible obstruction develops (blue line).

Furthermore, when a susceptible smoker stops smoking lung function does not significantly improve but the rate of FEV₁ loss returns to normal (green and brown line).

The concept of 'lung age' and the use of a formula for the calculation was first developed over 20 years ago.⁷² Morris and Temple developed a formula (Equation 1) based on reference linear regression equations permitting lung age estimation in terms of ventilatory function. This age can then be compared with the individual's chronological lung age.

Equation 1 Lung age calculation formula developed by Morris and Temple

Men

Lung age = $2.87 \times \text{height (in inches)} - (31.25 \times \text{observed FEV}_1 \text{ (litres)}) - 39.375$

Women

Lung age = $3.56 \times \text{height (in inches)} - (40 \times \text{observed FEV}_1 \text{ (litres)}) - 77.28$

Normal and abnormal groups determined by a respiratory health questionnaire and pulmonary function testing were used to compare the value of single and combination spirometric tests. The forced expiratory volume at one second (FEV₁) proved superior to any other single test or combination for best separation of the two groups and had the lowest standard error for estimated lung age. They speculated that both spirometry and estimated lung age calculation might be useful for motivating cessation of cigarette smoking.

Lung age is a way of conceptualizing this deterioration of function and a way of expressing lung damage rather than using mathematical concepts of percentage of expected value of FEV₁ for height, age and gender. For example

a 52-year-old man with 70% of normal function may have little or no symptoms and the individual may not appreciate the damage being done but if he is told that his lung age is 80 he may be more impressed. However, since that time no research has been published exploring the psychological impact of giving information to smokers in terms of lung age. Moreover, no research has tested the thesis that measurement of lung age could be effective in smoking cessation. The practical use of this concept is described using a graphic demonstration in Section 6.4 (p.189) on instruments and tests.

3.1.2 Does quitting reverse or arrest harm from smoking?

There is some evidence of improved lung function (FEV1) in the twelve months following cessation (by as much as 2%) but by far the most important benefit is the subsequent reduced rate of decline of lung function, calculated to be half the rate of decline compared to that in continuing smokers. Therefore it is well established that smokers who quit can benefit despite previous heavy smoking, advanced age, poor baseline function or airway hyper-responsiveness.⁷³

Much has been written about other benefits of smoking cessation. There are improvements to other systems of the body (in addition to the lungs) and also social and economic benefits for individuals and those who live with smokers.⁷⁴ Cardiovascular risk declines rapidly and significantly with smoking cessation.⁷⁵ Reduced mortality rates from cardiovascular disease in the US are partly attributable to reductions in smoking.⁷⁶ Within five years of quitting, women reduced their risk of coronary death and have a 20% reduction in smoking related lung cancer.⁷⁷ Further analysis and discussion of the social, economic and effects of passive smoking on families are beyond the remit of this thesis.

3.2 Why do people smoke and why is quitting so difficult?

Individual smokers may offer a multitude of reasons that they started, continue, or cannot stop smoking (or have relapsed). Quantitative research may give some information about the relative importance of some of these factors but qualitative research also has an important role to play in helping us understand the perspective of individuals. Reasons given by smokers for their behaviour may overemphasise their own perceptions of the cause and may depend on the circumstances in which they are questioned. Therefore, in this section I include evidence from a spectrum of research to give an overview of ideas about the causes of smoking and the difficulties in quitting.

3.2.1 Why do people start or stop smoking?

Most commonly people start smoking in their teens. The usual reason for starting is experimentation, which is motivated by psychosocial factors. In simple terms children start smoking to assert their perception of adulthood or as an aspect of rebellion. Smoking in children is most common if a teenager or child has close contact with smokers (parents, siblings or peers), has low self esteem, poor achievement in education, lives in socio-economic deprivation or attends a school where smoking is common.⁷⁸ Once the initial adverse effects of smoking have been overcome, the addiction to nicotine and habit set in to promote continued smoking. By the age of 20 years most smokers (80%) regret starting and will make repeated attempts to quit.⁷⁸

Every year approximately a third of adult smokers attempt to quit smoking but

typically relapse within days or weeks.⁷⁹ There are at least three distinct aspects to smoking cessation: the decision to quit, the attempt to stop and the maintenance of abstinence. Predictors of these different aspects are different.

A large prospective cohort study of smokers in four developed countries (Canada, Australia, USA and UK) including nearly 2000 people from the UK was published in 2006.⁸⁰ They used questions to measure intention to quit smoking and belief in their ability to succeed (see Appendix 3, p. 290). The questions of intention are almost identical to those used in the trans-theoretical model used to classify participants into stage of change into pre-contemplative, contemplative and action stages, but without any overt reference to that behaviour change model (see Section 4.4, p.116). Motivation to quit was assessed using questions about opinions of the health benefits of quitting and concerns over health and the impact on quality of life in the future from continued smoking.

Other questions were used to assess the scale of positive attitude over the time of the study and outcomes of quit attempts and successful cessation. They found that factors predictive of making a quit attempt included intention to quit, making a quit attempt in the previous year, longer duration of past quit attempts, less nicotine dependence, more negative attitudes about smoking and younger age. Lower levels of nicotine dependence were the main factor that predicted future cessation among those that made a quit attempt.

Their conclusion was that 'Intention to quit and other cognitive variables were

associated with quit attempts, but not cessation. Behavioural variables related to task difficulty, including measures of dependence, predicted both making attempts and their success'.⁸⁰ This aspect is highly relevant to the potential use of confronting individual smokers with the health consequences of their smoking, because an intervention may seem to motivate individuals towards change but in reality only change intentions without successfully changing behaviour. This problem is discussed further under the section on behaviour change models. It highlights that it is vital not to use 'intention' as a research outcome measure as it is not an accurate predictor or measure of actual behaviour change.

Reasons for quitting may be broadly divided into extrinsic and intrinsic (see Figure 2, p. 93).⁸¹ A prospective cross-sectional study of over 2500 North American smokers concluded that success was more likely if there were health concerns, higher levels of education and social pressures to quit.⁸² Concern about health is the most common reason patients give for quitting, and addiction is the most important barrier to quitting.⁸² Better education, social pressure, health provider advice, and formal cessation programs (but not over-the-counter devices), appear to increase the chances that smokers will quit.

Established disease attributable to smoking is also likely to lead to cessation.⁸³ However, many studies of smokers, demonstrate that a substantial proportion of smokers have co-morbidities caused by smoking, but nevertheless they are often resistant to changing behaviour and frequently have no immediate plans to stop smoking.⁸⁴ Extrinsic motivational reinforcement for reducing smoking in

COPD patients have been tried (e.g. lottery tickets for reductions in expired CO)⁸⁵ but with limited and temporary success.

The health benefits of quitting smoking have been well documented.⁸⁶⁻⁸⁸ Despite the evidence many individuals will resist change by expressing their enjoyment, denial of harm from smoking or simply on the basis of their rights of personal freedom. Personal rights come under pressure due to the wider implications of damage to others through passive smoking, harm to unborn children or harm within a household either directly or on the economic well-being of the family. Individual freedom has to be put in the context of another person's right to be free from harm. This is one of the reasons given for the recent ban in smoking in public places introduced in a number of countries. Moreover positive attitudes to smoking restrictions in public places are an independent predictor of the occurrence of smoke free homes.⁸⁹

3.2.2 Addiction

Addictions (as well as obsessions, and compulsions) relate to loss of voluntary control and getting trapped in a repeated cycle of self-destructive, harmful behaviour. Traditionally, scientists have confined the use of the term addiction to substance addiction. However, this has changed with the appreciation that the brain perceives various stimuli as a 'reward' that may be chemical or experiential.⁹⁰ Therefore, the term addiction cannot only apply to chemical substances but also to some activities (e.g. gambling).

Research has identified changes in neural circuits, which promote and continue the behaviour even in the absence of external chemical input. The changes are

mediated through the amounts of dopamine released whether the stimulus is behavioural or chemical.⁹¹ For example, as many as half of addicted gamblers demonstrate withdrawal symptoms similar to drug withdrawal and, like drug addicts, they are at risk of sudden relapse many years later. Therefore some chemicals (including nicotine) and some activities (gambling) appear to stimulate the brain's reward system. In addition, nicotine has been shown to alter the balance of neuron input, which increases the duration, and intensity of the pleasure from smoking.

‘Situated in a region of the brain called the ventral tegmental area (VTA), these reward-system neurons, called dopaminergic neurons, trigger release of the neurotransmitter dopamine (DA) in a nearby brain region called the nucleus accumbens (NAc). When nicotine attaches to these neurons they increase their activity, flooding the NAc with dopamine, which produces pleasure and a disposition to repeat the behaviours that led to it. That pleasure and disposition drive the process of addiction’.⁹¹

A detailed analysis of the neurochemistry and physiology of addiction is beyond the scope of this thesis, but it is important to determine where smoking lies in the spectrum of addictions and the characteristics that may be shared with other types of addiction and which may determine how people have difficulty with smoking cessation or relapse. On the spectrum of substance addiction nicotine is probably more addictive than cannabis or alcohol but is still in the modest range.⁹²

There are various schools of thought about the nature of dependence or addiction. It is apparent that not all addictive behaviours are pleasurable and not all pleasurable activities are addictive. There may be some delay from exposure to dependence and the progress of dependence can be extremely variable. Relapse is more likely during periods of boredom or crisis and in circumstances and situations which remind the person of their previous habit.⁹²

Approximately 80% of cigarette smokers can be classified as dependent according to measures of dependence defined by established tools (see Section 5.2, p. 149).⁹³ Compared to the treatment of other substance addiction, nicotine dependence has been investigated in a relatively large number of well-conducted randomised controlled trials. Most studies achieve a twelve-month abstinence rate of between 10 and 15% with a combination of pharmacological and psychological intervention (see Section 3.3.1, p. 62).

In the discussion about the psychology of behaviour change I include a number of theories from the social-cognition model. By definition these cognition models assume that a large part of behaviour and behaviour change is based on making conscious choices. Addiction can be seen, at least initially, as part of a rational informed choice. With the exception of malicious, criminal attempts at enforcing addiction to some drugs for illegal motives or profit it is reasonable to view the initial taking of drugs as a choice. Once on the path of taking drug, apparently many would see their consumption as a continuing decision process of weighing up the pros and cons of stopping or continuing. Therefore, some

observers deny the existence of addiction in the usual sense of an uncontrollable compulsion fuelled by psychological and physiological factors.⁹² Making reference to the attribution theory, they argue that drug takers continue to make choices, which confer benefits in the context of their life and circumstances. When confronted by authority (e.g. police or the medical establishment), drug takers may be content to assume the role of the addict who cannot control behaviour. According to this idea the addict's apparent lack of control needs to be excused or forgiven because they are not responsible for their behaviour. This translates into the addict's appeal of helplessness, the needs of the family in understanding why someone might harm themselves or their loved ones and those in positions of authority in helping to make sense of behaviour that is difficult to understand.

Assuming that addicts are making real choices some researchers have observed that addicts' so-called choices may change over time and be unstable even during a brief period (from one drink to the next). In choice theory, depending on personal preferences, desires, interests and their assessment of environmental constraints and opportunities they will weigh-up various alternatives, choose the best according to those preferences and beliefs, and then behave accordingly. However, it is important to note that the preferences may not be stable over time, and they do not have to be in line with social conventions and norms. They are dynamically inconsistent planners and they do not adhere to their original plans, but tend to give in to temptation.⁹⁴

Although I have presented the two extreme perspectives (addiction v. choice)

the choice theory rather ignores the compelling evidence for physiological craving and symptoms, which some consumers of tobacco experience. The theories of dynamic unstable choice and control does open a useful area of thought which permits us to view the choices of addicts, including many smoker, as real choice (and not simply driven by chemical forces) and therefore amenable to intervention or behavioural therapy.

3.2.3 Review of qualitative research

The value of qualitative research in the context of behavioural change is to establish the subjective reasons that people give about their continued smoking and difficulties with stopping or relapse after cessation. These studies have the potential value of supplying important information to help researchers develop understanding about smoking behaviour and difficulties and barriers with cessation and therefore the potential to help develop new and innovative ways to intervene.

Smokers express varying attitudes to advice from health professionals. Although many smokers expect advice when attending a health professional they do not believe that it will make a difference and either become annoyed or ignore it.⁹⁵ They may resent the interference when lifestyle advice is given.⁹⁶ The stages of change model of behaviour change shows that action oriented advice for those who are not ready to change is at best unhelpful, and could even entrench unhealthy behaviour. To make the most of opportunities for smoking intervention that arise in normal health care, it may be important to understand patients' perceptions of the acceptability of interventions they have received. Acknowledging the onset of actual harm may be a trigger to changed

behaviour and help quitting in those who are receptive.⁹⁷

Smokers often blame themselves for their smoking behaviour and believe that quitting is the responsibility of the individual and although they expect advice from professionals they do not have high expectations of benefit. Contact with professionals may simply enhance the feelings of guilt and alter health seeking behaviour.⁹⁵ Women from deprived economic circumstances experience guilt. Their guilt is related to worries over their own health and that of their children.⁹⁸ This concern may be justified as over a third of British children live in a household where at least one person smokes, and nearly 20,000 children under the age of five are admitted to hospital every year with illnesses resulting from passive smoking.⁹⁸

Smoking for many, especially the more disadvantaged, is a social and culturally ingrained behaviour.⁹⁸ Despite high levels of motivation to quit many of these women have lapsed into a hopeless frame of mind from repeated failure.⁹⁸ Descriptions of the power of smoking include words like 'craving', 'habit' and 'addiction' to describe their relationship with smoking.⁹⁹ Addiction is particularly used in the context of the first cigarette of the morning. For others in particular in socio-economic deprivation smoking may be a major pleasure in dismal life circumstances.⁹⁹ In contrast, whatever the social circumstances smoking after meals or with a drink are described as 'wanted' or 'pleasurable' or 'satisfying'.⁹⁹ Feelings of dependence on smoking not only cause hopelessness about the idea of quitting but also run in tandem with scepticism about the effectiveness of nicotine replacement therapy as this does not tackle the life circumstances that

they believe perpetuate their smoking.¹⁰⁰

The scientist's view of anxiety and smoking is that the symptoms of withdrawal are frequently interpreted as feelings of anxiety and a cigarette will relieve those symptoms, which therefore lead to the impression that smoking relieves anxiety.⁹⁸ Despite the fact that many people firmly believe smoking relieves their anxiety the data from international research contradicts that belief. Rather than helping smokers to relax, smoking probably increases anxiety disorders.¹⁰¹ Those with established COPD also continue to use cigarettes because they believe it will stop them getting bad tempered.⁹⁷ Stressful events or bereavement can be a trigger that causes relapse.⁹⁷

Beliefs and attitudes of certain groups of smokers may not be apparent to those outside certain cultural or religious groups. For example among some cultural groups in the UK smoking may be very common in middle-aged men (50% in middle aged Bangladeshi and Pakistani men in Newcastle) but it is rare and actually shameful among women.¹⁰² Among older men smoking is associated with maleness and socializing activity but is less acceptable and more hidden among young people.¹⁰²

While some cultures or religious groups accept or expect groups to smoke (middle aged men) or condemn certain groups (women and youth) the older smoker has seen a large shift in public opinion over their lifetime. Those with smoking related illness gained the habit in their youth when it was perfectly acceptable and a means of socializing, and now find themselves isolated and

smoking alone.¹⁰³ It is not known what factors promote smoking in later life especially when smoking related illness has already become established. In contrast, especially prior to the smoking ban in public places, smoking may be regarded as a sociable activity and perceived as demonstrating hospitality by not rejecting the smoker.¹⁰⁴ Sometimes social norms will support continued smoking rather than discourage it especially among young women in poor areas.¹⁰⁵

Self-perceptions may vary among different smokers. Some smokers are concerned about revealing their habit to others. Other smokers are not bothered and do not worry about the opinions of other people but will respect restrictions and avoid smoking near non-smokers.¹⁰⁶ A third typology of smoker – the adamant smoker – does not support restrictions and is less accommodating to other people.

Surveys in Britain demonstrate that the public appreciate that smoking is much more dangerous than other risks of death (e.g. from murder or road accidents).¹⁰⁷ All age groups underestimate the risk of death before the age of 70 years. The actual risk of death from smoking is about 250 deaths per 1000 smokers. However the median estimate (of death in smokers) given by those over 25 years of age was less than half the actual rate. There is little difference in perceptions of risk between groups of smokers, ex-smokers and never-smokers. There is no evidence that smokers deny the health risks of smoking or are less knowledgeable.¹⁰⁷ The notion of risk to self and to others has become very strong and has been discussed above when related to guilt and anxiety.

Many smokers from varying socio-economic backgrounds will try to restrict their smoking in circumstances where they might expose children to smoke. Most appreciate and accept the dangers of passive smoking.¹⁰⁴ Children are perceived as vulnerable and various self-imposed restrictions are reported by smokers to reduce exposure such as by opening windows, not smoking in cars. This is conceptualized as a moral identity of a caring parent or grandparent.¹⁰⁴

Qualitative studies in deprived areas of the UK have highlighted a number of barriers to use of smoking cessation services. Poor awareness of services and availability and effectiveness of interventions is a major barrier. Perceived cost of medication prevented many accessing services. Many fear the judgment of professionals and fear failure,¹⁰⁸ whilst others simply perceive a lack of support to help them stop smoking especially in deprived areas where smoking remains most common.¹⁰⁹

Across cultural groups in the UK, smokers experience failure with repeated attempts at cessation and believe that stress and fear of withdrawal symptoms prevent successful cessation. Often those wanting to quit find it difficult to resist the temptation to continue by on-going contact with those who smoke.¹¹⁰

Most smokers want to quit but feel unable to because of the importance of smoking in their daily routine and their addiction to nicotine. Even those with an established COPD diagnosis may attribute their symptoms to other factors (e.g. environment, work, pollution, age, fitness), often do not perceive any benefit in

trying to stop and attribute the death or deterioration of friends on their stopping smoking.⁹⁷ Social and family contacts of those with COPD may not be helpful. Some smokers even with established disease find that friends and family create barriers by offering cigarettes.⁹⁷

Many of these observational studies create a rich backdrop to smoking behaviour in different cultural and socio-economic groups. Even though no firm conclusions can be drawn and ideas cannot be generalised, the research provides a deeper understanding of some of the issues involved and may generate ideas for further research into effective interventions. In the context of this research project I am aware that the research population is not homogenous and that the individual response to the invitation to participate and the reaction to the intervention may be very varied.

3.3 Intervention trials in smoking cessation

The background quit rate without intervention is estimated to be between three and eight percent.¹¹¹ When assessing the rate of cessation it is important to consider the meaning of quitting, the population being studied and how the study group are recruited.¹¹²

In some studies, point prevalence at six or twelve months is measured. In addition the period of abstinence may be recorded and therefore cessation may not only be defined in terms of cessation at the time of data collection but also the length of time at that point prevalence that cessation has been successful (e.g. four weeks continuous abstinence). Therefore, there is a spectrum of

cessation, which does not simply equate with a simple dichotomy of being a smoker or non-smoker.

The relapse rate for those who have given up smoking is high. The National Health Service (U.K.) smoking cessation campaign measures quit rates at four weeks. Many research studies clearly state that any meaningful follow up of cessation should be done at twelve months to establish true quit rates for any given technique. For some people smoking or stopping is simply a matter of making a decision. For others it is a massive struggle with chemical addiction, habit, circumstances and environment. Some people who have quit smoking may remain dependent on nicotine replacement for a considerable length of time.^{107;107}

The factors that determine the transition from established smoker to established non-smoker are complex. The state of any individual should be seen as a place on a spectrum of behaviour. This change in behaviour will be discussed in more detail in the section about behaviour and change. A number of theories will be explored for different states of change and motivation.

Much of the vast literature on smoking cessation concentrate their attention on the various behavioural and pharmacological tools that have been used to aid people who have expressed a desire to quit smoking. The Cochrane database has multiple references to smoking. There are nearly 40 systematic reviews or meta-analyses. These include reviews of:

1. Acupuncture and related interventions for smoking cessation¹¹³

2. Antidepressants for smoking cessation¹¹⁴
3. Biomedical risk assessment as an aid for smoking cessation¹¹⁵
4. Community interventions for reducing smoking among adults¹¹⁶
5. Community pharmacy personnel interventions for smoking cessation¹¹⁷
6. Enhancing partner support to improve smoking cessation¹¹⁸
7. Exercise interventions for smoking cessation¹¹⁹
8. Group behaviour therapy programmes for smoking cessation¹²⁰
9. Individual behavioural counselling for smoking cessation¹²¹
10. Interventions for promoting smoking cessation during pregnancy¹³
11. Nicotine replacement therapy for smoking cessation¹²²
12. Nursing interventions for smoking cessation¹²³
13. Physician advice for smoking cessation¹²⁴
14. Relapse prevention interventions for smoking cessation¹²⁵
15. Self-help interventions for smoking cessation¹²⁶
16. Smoking cessation for chronic obstructive pulmonary disease⁵⁷
17. Hypnotherapy for smoking cessation¹²⁷

A detailed analysis and comparison of the different aspects of smoking research is beyond the scope of this thesis. However it is useful to have an overview of the breadth of interventions that have been tried in primary care in order to put this research thesis into perspective.

As the main emphasis of smoking cessation intervention in the NHS are psychological support (counselling) and nicotine replacement therapy these are included in the literature review. This research project did not set out to directly

compare the efficacy of NRT or counselling with the lung age intervention but participants were free to access local smoking cessation facilities (although only a small minority of participants in this research project reported using NRT in their quit attempt after our intervention). It is also highly likely that the participant populations are very different due to recruitment methods, the information given and inclusion and exclusion criteria. Therefore, it is important to consider how the different types of intervention being investigated might attract different participants, which could influence the relative success of the interventions.

Despite those reservations, it is worth putting our research into the context of some of those other well-established and widely researched interventions and to consider the relative usefulness of the interventions using nicotine replacement therapy or counselling services in research and in this thesis. Therefore I will briefly outline some of the key studies that have shaped opinion and practice within the United Kingdom so that some of these comparisons can be made and will I discuss the other aspects in a later section (see Section 8.2, p. 207).

3.3.1 Smoking cessation trials

Nicotine replacement therapy (NRT)

Nicotine replacement therapy (NRT) has been a key area of research in smoking cessation for more than 30 years. There is general agreement in most research into NRT that it works for a proportion of smokers and in many different developed countries and contexts but that relapse rates are high.

Early trials of the efficacy of NRT were not promising. One of the earliest published trials (1980) of NRT in the United Kingdom tested the effect of NRT chewing gum against placebo gum in 200 volunteers who wanted to stop smoking, mainly recruited from newspaper and workplace advertising.¹²⁸ After the treatment, initially about a third of both the intervention and placebo groups had quit but by six months most of them had relapsed and the differences with control were not significant. The numbers were too small for statistical assessment. No psychological measure of motivation or nicotine dependence was reported but all volunteers had enrolled 'wanting to stop'. Despite voluntary participation with the stated aim of quitting these results were disappointing. This early study would have cast doubt on the efficacy of this therapy but larger studies were needed to establish the role of nicotine replacement therapy

It might be assumed that highly motivated smokers given the opportunity for support and help with symptoms of nicotine withdrawal would do well. A moderate sized study conducted in primary care in Nebraska, USA, used NRT patches in well motivated smokers consuming at least 20 cigarettes per day.¹²⁹ Nearly 400 smokers with high levels of motivation to stop were randomised into a double blind placebo controlled trial in 20 primary care clinics. Motivation was assessed using an eleven-point scale and confirmed to be high and evenly distributed between the groups. They were all given brief counselling on two occasions during the study. The twelve-month abstinence rates for the active and placebo patch groups were 14.7% and 8.7%. The over 45 year olds and those with higher nicotine dependence and those who spontaneously volunteered did better with NRT compared to the equivalent subgroup using

placebo, although the study was not powered to detect significant differences in those subgroups.¹²⁹ Considering that these smokers were supposedly highly motivated to quit the quit rates are not impressive but nevertheless are in keeping with other studies of NRT improving rates by about 50%. Duration of counselling, older age and higher levels of dependence were in favour of better quit rates compared to placebo.

In a primary care study in Oxfordshire, written invitations were sent to those aged 15 to 65 on 19 practice lists. Others smokers were recruited through contact with recipients of the letters. An average of 32% of the 15-65 year olds in these practices were invited. In those days (1994) computer registers of smokers would have been less available or accurate and the authors did not report what proportion of smokers responded to the invitation. Nevertheless, this randomised double blind trial of nearly 1700 smokers with confirmed cessation, using carbon monoxide and saliva cotinine testing, resulted in rates of cessation of 10.8% in the NRT patch group and 7.7 % in the placebo group continuously from 3 months to 12 months.¹³⁰

Other studies in the UK have produced similar results over a twelve-month period. A multi-centre randomised double blind placebo controlled primary care study recruited 1200 smokers between 20 and 60 years of age who were motivated to quit. Approximately 70% of participants wanted 'very strongly' to stop smoking altogether.^{131;132} At one year the nicotine patch group achieved 9.3% continuous abstinence compared to 5% in the placebo group. An important deficiency of the generalisability of this study to primary care was the

deliberate exclusion of those who would have most to gain from quitting notably those with heart disease, hypertension or diabetes. They also failed to include clear methodological details of their scales for measuring motivation and dependence.

Attempts have been made to compare the relative success of using different strengths of NRT for smokers with different levels of nicotine dependence as scored by the modified Fagerstrom score and the heaviness of smoking index (HSI).¹³³ Garvey et al. recruited just over 600 volunteers through newspaper advertising in the Boston area and randomised them to receive placebo or one of two different strengths of NRT gum.¹³³ Brief counselling was on offer for about ten minutes at each contact. After twelve months the abstinence rates were 6.4% in the placebo, and up to 13.4% in those using NRT. Low dependence smokers did better with placebo than high dependence smokers as one would expect but gum strength did not make a significant difference to the success of high compared with lower dependent smokers. Therefore gum users doubled their quit rate over-all compared to placebo. Again levels of initial motivation were measured as high on the same scale as the Nebraska study by Daughton et al.¹²⁹ (described above) and recruitment method attracted those with a prior desire and confidence of quitting success (average confidence of success >7 on a scale up to 10).

None of the studies above included data about co-morbidity and their recruitment methods tended to exclude those at most risk from continued smoking (e.g. cardiac disease). One can therefore conclude that healthy

volunteers who are well motivated are a group who can (at most) double their chances of quitting compared to their respective controls treated with placebo. It is important to know how well those with serious co-morbidity would respond to intervention to stop smoking. Smoking may cause serious disease or cause acceleration of complications of other diseases (like diabetes)

A Welsh study specifically recruited those who have smoking related disease. This group already are at special risk of exacerbating their primary disease or of secondary events and therefore likely to be well versed in the dangers of smoking. Therefore they are likely to represent a group who struggle with their habit and are resistant to change.¹³⁴ In a twelve-month period the clinicians referred over 400 high-risk patients to see the smoking counselor. All had recently been admitted with smoking related diseases included those with chronic obstructive pulmonary disease, coronary heart disease and vascular disease.¹³⁴ Of these nearly half either did not attend or refused to be included in the trial. They were randomised by month of entry to a control group, which received monthly counseling support or an intervention, which involved counseling and NRT. At the end of twelve months quit rates in both groups was the same at around 14%. This outcome in this special group of high-risk individuals can be interpreted in a variety of ways. It could be viewed a success that the extra support enabled 14% to quit for 12 months or that nicotine replacement is not the key for these smokers or that even if health is overtly being harmed the majority of smokers are unable to quit. It is unfortunate that this study did not include any psychological measure of motivation or self-efficacy. This prospect that pre-existing smoking related disease does not seem

to be a major stimulus to change has major (negative) implications for the intervention presented in this thesis and for other research that concentrates on the impact of confronting smokers with the real consequences of their habit.

Two very important findings have emerged from more recent reviews of the data. Firstly long-term quit rates are most important but few studies continue beyond one year. Those that have done long term follow up have demonstrated continued attrition of the benefits.¹³⁵ Secondly, randomised controlled trials often exclude the most vulnerable patients. They select patients with lower levels of co-morbidity or have exclusions that render the results non-generalisable. Therefore those participating are not representative of patients in normal general practice. Trial participants are generally healthier and are likely to be more motivated to quit smoking.¹³⁶

The implications for practice of the research on NRT are neatly summarised in NICE Guidance.¹³⁷ Therefore they will not be reproduced in this thesis except for the following excerpt:

‘All of the commercially available forms of nicotine replacement therapy (NRT), i.e. gum, transdermal patch, nasal spray, inhaler, lozenge and sublingual tablet, are effective as part of a strategy to promote smoking cessation. They increase the rate of long-term quitting by approximately 50% to 70% regardless of setting. These conclusions apply to smokers who are motivated to quit and who have high levels of nicotine dependence. There is little evidence about the role of NRT for individuals smoking less than 10 to 15 cigarettes a day’.

Smoking cessation, COPD and NRT

As outlined in the previous section, only a minority of studies of NRT have deliberately targeted high-risk groups and those with smoking related co-morbidity. With respect to this research thesis on the impact of lung age estimation, the group of people who already know they have pre-existing lung damage are of particular interest.

One systematic review (2004) attempted to identify studies that targeted those with chronic obstructive pulmonary disease.¹³⁸ The five studies that remained after their initial search were a heterogeneous group with a variety of interventions. Three of the studies recruited less than 60 participants each and two of them did not isolate the effect of NRT. One study used the concept of 'smoker's lung' without any other form of intervention or pharmacotherapy to promote behaviour change but was small, suffered contamination between groups and it could not produce meaningful statistical differences.¹³⁹

Finally they included a large randomised clinical trial (ten centre and over 5000 patients) in the US and Canada but it was not primarily designed to study the use of smoking cessation interventions.¹⁴⁰ Their main objective was to determine whether a program incorporating smoking intervention and use of an inhaled bronchodilator can slow the rate of decline in forced expiratory volume in 1 second (FEV1) in smokers aged 35 to 60 years who have mild obstructive pulmonary disease. However they did randomize their sample to three groups; a smoking intervention plus bronchodilator, smoking intervention plus placebo,

or no intervention. They concluded that the success of smoking interventions in this group of respiratory patients is low despite the high risk of progression to more severe COPD.¹⁴⁰ They stated that this is consistent with the commonly held view that continued smoking in the presence of established COPD is an indication that these patients are particularly addicted and would have already quit if they were likely to. This rather defeatist view is a real barrier to progress when it comes to research and treatment of those with COPD at any stage. There is almost a hopeless resignation by authors to the inevitability of negative attitudes and lack of changed behaviour. Generally the paucity of studies of smoking cessation and COPD and the poor methodological quality and size of studies that have been done are a further reflection of negative attitudes about those with COPD.

Finally, a recent update of a Cochrane review (2008) analysed 111 studies which tested the efficacy of one or more types of NRT.¹⁴¹ All the studies in the analysis compared the effect of NRT to placebo or no NRT. Definitions of abstinence in those studies varied widely with three quarters of them reporting sustained abstinence or repeated point prevalence. Some were self-reported but most had some type of biochemical verification. The most common form of validation was measurement of carbon monoxide in expired air. Validation of cessation by cotinine (in blood saliva or urine) was included in 27 trials. They produced evidence from trials, which included data from a total of over 40,000 participants. They concluded that offering NRT to dependent smokers who are prepared to try to quit increases the chance of success irrespective of the method of delivery of NRT. The absolute effect of NRT depends on a number of

different factors evident in different studies. Without any pharmacotherapy or support quit rates over twelve months are about 3-5%. Using NRT can increase successful quit rates by 2-3% (NNT 33-50) but where study populations are primed to better baseline quit rates (by better predictors of success or intense behavioural support) the rates may be improved from 15% in controls by 8% (NNT 12). In other word there is a 50% improvement from the baseline wherever that happens to be.

In conclusion, although nicotine replacement therapy (NRT) is not an integral part of the intervention in this research project, it is likely that quit rates and cost comparisons will be made. It is fair to say that NRT works well for those who use them as part of a supervised smoking cessation programme. This presupposes that people are motivated to attend these programmes and comply with taking medication in the prescribed manner. However, they should not be used or offered to those who have little intention to give up smoking.

Motivational interviewing

Intensive counselling and even brief verbal intervention in primary care by clinicians will increase the likelihood that someone will give up smoking. Attempting to quit smoking involves two major components. The two distinct parts of the quitting process are the initiation of an attempt and then maintaining cessation.²⁷ Although the majority of smokers would like to quit there are some who need motivating to change. In a large survey in four developed countries, it was found that the majority of smokers would like to quit.⁸⁰ It is important to motivate smokers to act upon these desires and to encourage patients to stop as quickly and as early as possible.

Motivational interviewing (MI) attempts to 'overcome the ambivalence that keeps many people from making desired changes in their lives'.¹⁴² Proponents of the technique conceptualize MI as occurring in two phases with two overlapping goals. The first phase involves building intrinsic motivation. In practice this is usually the actions of a health professional by highlighting the problem at every opportunity in a way that is not going to jeopardize the client-professional relationship and that is appropriate to the stage of motivation. With reference to stage it is important to note that motivational interviewing draws heavily on the assumption that the trans-theoretical model of change is a well-established, accepted and sound theory. Phase one of MI is regarded as non-confrontational and uses open questioning with reflective listening techniques similar to many forms of counselling.

The second phase of MI involves strengthening the commitment to change and developing a plan to accomplish the transition.¹⁴² The second phase of MI uses confidence-building techniques. This avoids the idea that the professional can 'sort the problem out' and draws the client away from negative thoughts about their own ability to make plans to change and to build problem solving skills. For example asking:

'What problems do you foresee, and how might you deal with them?'

The technique and type of questioning could be conceptualized in a number of cognitive and social cognitive theories. This is evident by the use of open questions about confidence to change that might be called self-efficacy, and

personal strengths as well as social support and previous experience of the smoker.

Most of the evidence for the utility of MI in changing behaviour comes from the realms of problems with alcohol, gambling or physical activity.¹⁴² Part of the difficulty assessing the content and quality of trials is the use of an ill-defined intervention, which may or may not be reproducible by different therapists. For example in assessing the results of various studies which purport to use MI it is necessary to understand what the researchers actually did, for how long and with what training. Essential components often include feedback to a person on their individual results derived from standard assessments. In fact most research studies purporting to be testing the efficacy of MI use adaptations of motivational interviewing (called AMI) which contain an element of feedback carried out by non specialists.¹⁴³

There have been only two controlled clinical trials of motivational interviewing in smokers. Only one of these investigated smoking in adults and was large enough and had sufficiently long follow up to be worth considering here.²⁶ The main outcomes were behavioural measures and readiness for change at six months. Overall the results were disappointing and there were no significant differences in abstinence, reduction in cigarettes smoked or overall quit rates. However there was a movement towards a more advanced stage of change.²⁶ As discussed before, a change in stage is not a reliable or useful primary outcome for trials, as it does not equate to behaviour change.

So far the research on motivational interviewing has been inconclusive due to low power, inadequate follow-up and problems with consistent delivery of the intervention.

3.4 Biomedical markers

A possible strategy for improving smoking cessation rates could be giving feedback on the biomedical or potential future effects of smoking in a more personalised way in comparison to the broad generalised public health message of 'smoking is harmful'. The simple facts are that a minority of smokers will get cardiovascular disease (15%)¹⁴⁴ and the majority of smokers will not get chronic obstructive lung disease (75%).⁵

Although 'smoking kills' is written on many packets of cigarettes only a quarter of smokers will die of diseases attributable to their habit.^{16;107} Although smoking is the major cause of lung cancer and smoking cessation is recommended, large trials indicate that the lag time for a beneficial effect of smoking cessation on lung cancer may be as long as 20 years.¹⁴⁵

A Cochrane review has shown that community based health messages about smoking have been ineffective.¹¹⁶ However, the concept of using personalised risk as a method of encouraging behaviour change has some merit and has been investigated in a number of different contexts. Biomarkers can be broadly classified into three types as indicators of:

1. Exposure to harmful substances e.g. the presence of cotinine in

smokers' urine indicating exposure to harmful chemicals

2. Damage caused by exposure to harmful substances e.g. abnormal angiograms in smokers

3. Susceptibility to disease as a result of the marker e.g. the presence of genetic markers indicating an increased risk of lung cancer.

The factors that trigger and sustain changes in health behaviour are difficult to evaluate. A number of different models of behaviour change have been used to try to predict or explain the actions of individuals. Some of them combine several aspects that individuals consider when making decisions. For example their perception of personal disease susceptibility, the severity of that disease according to their understanding and experience and an analysis of the benefits or risks of changing behaviour may influence an individual (see Section 4, p. 89). Motivational interviewing incorporates elements of these three factors to treat alcohol, opiate and cigarette use.¹⁴⁶

3.4.1 Non-systematic review of biomarkers

McClure has published a non-systematic overview of twelve studies using feedback on individual's biomarkers.¹⁴⁷ Some studies used multiple markers ranging from genetic markers for lung cancer risk, through to urinary, blood or breathed air markers of smoking. Their conclusion was that the successful use of biomarkers to alter attitudes of those with unhealthy behaviour probably depend on several interrelated factors-

- How the information is conveyed.
- If the information is understood and accepted.

- Understanding of the purpose of the marker and what is actually being tested and how it relates to their behaviour and health.
- Comprehension can be enhanced by use of written handouts and graphic displays of the biomarker values.
- Repeated measurement and reinforcement of the message.
- How the message is conveyed in terms of harm or benefit. According to prospect theory people respond better to the prospect of potential gain rather than the messages about costs and disadvantages.

Weaknesses of review

Unfortunately the evidence used for the above conclusions have some major flaws. Apart from the fact that that there was no clear search strategy, a number of the research papers that they used in their review had serious methodological shortcomings.

The review claimed that 'biomarker feedback may enhance the likelihood of cessation because a trend for increased abstinence was found in three randomised trials'.^{36;148} However, one of the three papers quoted and reputed to be by Hoffman 1998 was never published (claimed to be in Behavioral Medicine Journal). The other two trials had serious methodological shortcomings. For example:

1. Risser and Belcher studied 90 smokers in a general screening clinic.³⁶ They were randomised to receive education alone or education plus an additional

motivational intervention that contained immediate feedback about the smoker's exhaled carbon monoxide (CO) values, spirometry results, and pulmonary symptoms. Including all patients not successfully contacted at follow and assuming they were continuing to smoke, the self reported quit rates were 20% vs. 7% ($p = 0.06$) in intervention and control groups respectively, and therefore not significant. The major methodological problems with this study were the absence of a power calculation, small numbers in the study, no clear randomisation procedure, and the lack of generalisability as the study was predominantly in U.S. male veterans (96%).

2. Walker and Franzini recruited 64 smokers, by media advertising and financial incentive, into this randomised controlled trial study comparing two therapies.¹⁴⁸

The intervention group had exhaled carbon monoxide measurement and spirometry feedback. Half of them also had taste satiation (TS) or focused smoking (FS) (in 50%) and voluntary booster sessions. The control group did not have spirometry or carbon monoxide measurements but otherwise were treated the same. The setting was a stop-smoking clinic in the United States. The method of randomisation was not explained. Only 64 agreed to take part and the 2 x 2 groups therefore contained very small numbers. Follow up at six months measured quit attempts and biochemical validation of non-smoking. The main problems with this study were the lack of a power calculation, small numbers of participants divided into very small groups for the complex 2 x 2 study which resulted in a lack of data for analysis which prevents any meaningful results or conclusions. Not surprisingly no statistical analysis was given.

Four other studies included in the review were trying to simultaneously test many different components so that the effect of the biomarker could not be identified or isolated. In addition they were not randomised controlled trials.

A study by Li et al. tested the hypothesis (one of three hypotheses) that male asbestos-exposed workers who had abnormal pulmonary function tests would be more likely to respond to counselling by a physician to stop smoking than those with normal results.¹⁴⁹ From 1231 men recruited only 579 were successfully followed up at 11 months. The participants were not properly randomised. Those with abnormal pulmonary function test result (PFT) were randomised to get minimal counselling or detailed prolonged counselling on the basis of their results. Only 47 had abnormal pulmonary function and contrary to the protocol one of the research physicians did not comply with the counselling protocol and the groups had to be re-constituted.

Despite the deficiencies of the study the researchers were surprised to find that prolonged cessation rates of abnormal PFT subjects were not different from those of subjects with normal PFTs. In fact they found that those with normal results were much more likely to give up 4.3% v 9.5%. Neither a power calculation nor confidence intervals were given. Questionnaire data showed that nearly all subjects perceived a link between smoking and lung disease (90%) and had previously tried to quit (77%). Although the study was fatally flawed by methodological problems it does raise the interesting fact that one

cannot assume that bad results of lung function and lots of counseling will yield better quit results than good results and similar or less counselling and information.

Overall, I conclude that the review by McClure is useful in highlighting some of the published studies but disappointing in that the quality of studies means that no real conclusions can be drawn. However on the positive side some ideas have been generated about what might influence smokers and is a source of lessons for future research and interventions.

3.4.2 Critical appraisal of Cochrane systematic review (2005)

A detailed and comprehensive review by Bize et al was published in October 2005, twelve months after the start of the Step2quit RCT and more than two years after the initial development of the research proposal.¹¹⁵ As this is such an important and relevant review for this thesis I present a critical appraisal of this review below.

In the context of my research proposal, the studies of particular interest are those in which markers of lung damage have been used to promote smoking cessation. It is important to note that none of the studies selected by the Cochrane review used lung age as tool for measuring or explaining the effects of smoking on the lungs. A summary of some key aspects of the different studies outlined in the Cochrane review are contained in the Appendix 2 (p. 287) along with the key flaws in methodology.

The systematic review by Bize et al will be critically appraised according to the following headings.¹⁵⁰

1. Has the review addressed an important clinical question?
2. Have the reviewers made a thorough search and explored other sources?
3. Was methodological quality assessed and trials weighted?
4. How sensitive are the results to the way the review was done?
5. Have the numerical results been interpreted with common sense and due regard to broader aspects?

Has the review addressed an important clinical question?

Antismoking campaigns and public health bodies have used blunt and occasional graphic messages to inform, alert and scare smokers for many years. Very little evidence is available that these are effective in reducing smoking but the display of general aversive information has effectively taken responsibility about harm away from the producers of cigarettes and governments and firmly places the responsibility onto the individual.¹¹⁶

Whilst the public health messages and other strategies to improve health education and reduce smoking should continue it is important to review the messages given to individuals and the most effect way of giving that information. The Cochrane collaboration correctly concluded that this is an important area of study with the following central focused question:

‘Does feedback on personal characteristics indicating effects of smoking, or susceptibility to smoking-related illness, increases rates

of smoking cessation?’

And the following objectives:

- To determine the efficacy of biomedical risk assessment provided in addition to various levels of counselling, as a contributing aid to smoking cessation.
- Are multiple types of measurement (e.g. spirometry and exhaled CO measurement used together) more effective for smoking cessation than single forms of measurement?

Have the reviewers made a thorough search and other sources explored?

They searched the Cochrane Collaboration Tobacco Addiction Group Specialized Register, Cochrane Central Register of Controlled Trials (CENTRAL), MEDLINE (1966 to 2004), and EMBASE (1980 to 2004). They combined methodological terms with terms related to smoking cessation counselling and biomedical measurements in a thorough, explicit and systematic way. They were very strict and rigid in their choice of trials for further analysis. The pros and cons of this will be discussed below. They included only RCTs of smoking cessation trials with interventions based on a biological test aimed to improve motivation. They excluded trials in which the effect of biomedical test was confounded by the use of other components in the intervention group. From 4049 retrieved references, they selected 170 for full text assessment. The researchers give a very clear indication of their intention to include only randomised controlled studies, which display rigorous quantitative methodology. For this reason out of the original 170 selected abstracts only eight trials met the inclusion criteria. In conclusion, the review

conducted a thorough search for sources in keeping with Cochrane principles.

Was methodological quality assessed and trials weighted?

The trials were assessed according to rigorous criteria. Studies were excluded if they were not randomised trials or if they did not have feedback on biological measurements of some aspect of the smoker's health. If the intervention strategy was not clear-cut or the period of follow up was deemed inadequate (six months) they were excluded. Furthermore the quality of each trial was assessed using transparent pre-determined criteria. Despite their strict criteria, among the eight studies that were included in their final list for detailed analysis, there were plenty of flaws in the methodology. For example only one of the eight trials reported an adequate randomisation procedure.¹⁵¹

How sensitive are the results to the way the review was done?

If anything the methodology of this systematic review is so rigorous and exclusive that no other conclusion could be reached. It is feasible that a more inclusive approach may have found some merit in the use of biomedical markers but would have carried less weight. The authors state that they used other studies (non-RCTs) for background information but not for any objective analysis of effectiveness. This approach is in keeping with the Cochrane principles and the SIGN hierarchy of evidence. However if cross sectional studies and those which concentrate on qualitative analysis is ignored important information and insights from other studies may be overlooked. However, these issues will be discussed more under the section 3.4.3 below about trials that have used interventions confronting smokers with their lung damage to aid

behaviour change.

Have the numerical results been interpreted with common sense and due regard to broader aspects?

As all the included studies were randomised controlled trials Bize et al converted the results into odds ratios for smoking cessation up to the longest recorded follow-up time (six to twelve months) between intervention and control groups, with their 95% confidence intervals. Mostly they were unable to combine results because the interventions were too diverse. In the two studies they were able to combine for a mini meta-analysis the effects were in opposing directions and more or less cancelled each other.^{152;153} They questioned the external validity of the only study with a statistically significant positive odds ratio as the sample was made up predominantly of male light smokers in a developing country (average 10 to 12 cigarettes a day).¹⁵⁴ Moreover, from the perspective of this thesis that study had little relevance as the intervention was about confrontation with evidence of arterial damage rather than lung damage.

However the reviewers could not say with certainty that there is no effect from using biological markers. Several of the studies had odds ratios greater than one, which therefore favoured the intervention group, but with confidence intervals that straddled zero and therefore reduced the likelihood that the real result was positive. Any effect may be hidden by a serious lack of power to detect small but significant changes in smoking behaviour. Remarkably, only one of the studies included in their final selection had documented a power calculation estimation of sample size prior to recruitment.¹⁵⁵ Moreover, that

estimate was based on a quit rate of 25% in the intervention group and 10% in the control group. In the light of other interventions for smoking cessation this was probably a rather optimistic estimate of the possible effect of intervention.

Another possible explanation for the absence of effectiveness of biomedical risk assessment could be the potentially counterproductive effect of communicating normal results to smokers. Only one of the studies included in the analysis provided some insight about smoking cessation rates according to spirometric test results. Sippel did not find any correlation between smoking cessation and abnormal spirometry results.¹⁵⁵ These particular questions, and the best way to handle and communicate normal results, remained unanswered.

It is clear that there are some serious methodological shortcomings in the studies that were included in the review. Even if the results of the statistical analysis had pointed towards a useful effect of the intervention these methodology problems would have created serious doubt about their usefulness and generalisability.

Therefore it is possible that with better methodology with improved numbers of participants, adequate length of follow up and experimental rigour these interventions could be re-investigated for their possible effectiveness. In these circumstances isolating a small but critical improvement to smoking cessation or uptake of smoking cessation treatments would be more likely.

Elsewhere I have outlined the study by Etter et al. (see Section 4.7, p. 141) of

tailored information given to smokers according to personal data from a pre-intervention questionnaire.¹⁵⁶ It seems clear that the effect of such brief interventions will be limited, subtle but possibly important especially if clinicians are to 'capture' those smokers who would not normally attend a smoking intervention because they are not (yet) motivated to do so.

It is already fairly clear what support and pharmacological treatments help smokers to quit when they are seriously intending to do so. The systematic review did not reveal strategies to help the reluctant but damaged individuals or to establish a role for the routine use of biomarkers in smoking cessation policy. However, the authors concluded that due to a lack of good quality evidence they could not make any definitive statements about the effectiveness of biomedical risk assessment as an aid for smoking cessation.

3.4.3 Smoking cessation in the presence of lung damage

Several researchers have tried to find an association of abnormal lung function with improvement in cessation rates. A large non-randomised study of 4500 Polish men and women smokers found that those with lung damage from smoking were more likely to respond to stop smoking after being confronted by information about their lung damage compared to those who had no detectable damage at baseline.¹⁵⁷ At the time of screening all subjects received simple smoking cessation advice. Those with abnormal lung function were told that they had COPD and that smoking cessation would halt rapid progression of their lung disease. They were shown a graph adapted from Fletcher and Peto's classic diagram demonstrating deterioration with age in susceptible smokers (see Figure 1, p. 44),⁶ and their FEV1 was superimposed on the diagram

showing the expected future decline and the benefit of stopping. They were strongly encouraged to stop smoking and given a booklet about health risks of smoking and benefits and methods of stopping.

The validated smoking cessation rate in those with airway obstruction was 16.3% compared with 12.0% in those with normal spirometric parameters. Although they did not use lung age as a concept to explain their results use the idea of showing a graphic and giving individualized advice is a similar idea. The study has a number of shortcomings. The control group quit-rate in those with normal lung function is considerably higher than expected which lead to some doubts about how generalisable the results can be. The participants were allocated and not randomised. The characteristics of the two groups are therefore very different and other unidentified confounders may be influencing the results. Of note the measure of pack-years, age and of course (by definition) percent of predicted FEV1 was different. Success in smoking cessation was associated with lower lung function, lower nicotine dependence, and lower tobacco exposure. This study would not have been included in the Cochrane analysis but adds some further evidence of a possible use of graphic displays and explaining deteriorating lung function to smokers. The implication from the Bednarek study is that individuals can be presented with understandable individualized, graphic information that creates concerns about the future deterioration of function, which can translate into possible concern about health, negative thoughts about smoking, and action to quit.

Conceptualizing and operationalising (measuring) these psychological factors

have proved more difficult and have not thrown more light on any possible mechanism. In a small, randomised controlled trial of 124 smokers the perceived risks and feedback about lung age did not correlate with desire to quit.⁵⁶ This study also would not have been included in the Cochrane review above because the primary outcome measure was 'desire to stop smoking' not smoking cessation. Furthermore, the participants were college students with a mean age of 20 years and therefore not representative of the usual group of smokers in the community, and the numbers were too small for any statistically significant conclusions to be made. One surprising feature was that 75% of the students had a lung age greater than chronological age (the mean lung age was 35). Most previous research indicates that 20 pack years of smoking are required before lung damage is detectable.³² This discrepancy casts doubt on the reliability of the testing.

Several other studies published since the Cochrane review of 2005 have produced mixed results. A prospective primary care study in Sweden used annual spirometry and brief advice over an impressive three years of follow up.¹⁵⁸ They identified those with COPD and compared them to a group of smokers with normal lung function. This was not a randomised controlled trial and the groups were different by definition. The authors also suspected higher motivation due to the recruitment process of advertising a free lung test for smokers between 40 and 55 years. The abstinence rates were significantly higher in the smokers with COPD than in smokers with normal lung function. Smoking cessation rates among smokers with normal lung function did not increase with increasing number of follow-ups. Despite its limitations the 3-year

cessation rates of 25% in the COPD group (v 9% in those with normal lung function, $p < 0.001$)^f give some support to the value of annual spirometry and discussions about abnormal lung function. They concluded that smokers diagnosed with COPD stopped smoking significantly more often than those with normal lung function.¹⁵⁸ Two major weaknesses of the study cast some doubt over the results. First the smokers were not randomised and therefore there may have been some unknown confounding variables and secondly the reliability of their impressive quit rates is questionable as these were self-reported and not validated by biochemical testing.

Finally, the study that comes nearest in methodology to the lung age study was a randomised controlled trial in primary care in Belgium.¹⁵⁹ One major difference was that their study excluded those in pre-contemplative and contemplative groups. They selected only preparation and action stage smokers who were randomised by a toss of a coin and used spirometry results as part of the intervention. All 221 participants tried to quit, a random sample were confronted with spirometry results. They report that they found no significant difference in those with normal or abnormal results or those with spirometry versus no spirometry. Their interpretation is based on 24 month quit rates of 19% v 14% but no detailed statistical analysis was presented. They also did not include a power calculation and failed to document levels of co-morbidity or specify exclusion criteria (except stage of change). In comparison to long-term quit rates in other studies their rates seemed fairly respectable. However, I believe their study is inconclusive and incomparable with the lung age study for several

^f No CI reported

important reasons. First the study was not sufficiently powered to detect a difference (a five percent difference in smoking rates was said to be non-significant) and secondly their sample was highly selected as far as motivation to change.

In summary, my overall conclusions from the research published since 2005 Cochrane meta-analysis is that the jury is still out about the use of confronting smokers with their spirometry results however that is done. More and better quality research is required to establish the role of using spirometry in screening and in motivating all smokers towards behaviour change.

4 Literature review 2: Theories of behaviour change

4.1 Introduction to behaviour theories

It is beyond the scope of this thesis to cover all behaviour theories. Out of hundreds of possible theories of behaviour change I have chosen to focus on several for detailed discussion. They are the theory of planned behaviour (TPB), catastrophe (chaos or complexity) theory (CCC), the trans-theoretical model (TTM), and the health belief model (HBM).

My reasons for choosing these particular theories for further discussion are mostly pragmatic. As a non-psychologist I take an outsider's view and cannot be exhaustive in covering the discipline of behavioural psychology. I have therefore chosen theories that:

- Are frequently cited in the health research literature in the context of smoking, addiction and health related behaviour change;
- Appear to have some significant differences to each other, and therefore provide an opportunity to explore the range of ideas and constructs used in theories;
- Seem to have significant overlap in constructs and terminology, which I compare and contrast.

For each of the selected theories, I outline the fundamental structure, assumptions, how they work, the terminology, some research background, and

the usefulness of each model in relation to smoking.

4.1.1 What are theories of behaviour for?

The development of different theories in western medical practice reflects the positivist approach to most biomedical research and intervention. Positivism assumes that there exist constant, replicable and testable facts. Positivism and the scientific method are inextricably intertwined. The scientific method depends upon gathering observable, empirical measurable evidence. Even though many research articles are concerned with facts and results and may not pay attention to an underlying theory there is an assumption (even if unacknowledged) that such fundamental rules underpin modern scientific enquiry.¹⁶⁰

Most of the models of behaviour indicate that their primary objective is to predict behaviour.¹⁶¹ Stepping back one stage further it is clear that there are two basic assumptions that justify the study of health behaviours. First, that a large proportion of morbidity and mortality (particularly in industrialised countries) is a consequence of certain behaviour and second, that these behaviours are amenable to manipulation and change.

Another important objective of developing models is to explain observed behaviour in such a way that the knowledge can then be used to develop new strategies to promote desired behaviour change using that information as the underpinning foundation for new interventions. Finally, although cognitive theories are mainly used to predict behaviour they may have a sensitising role, generate ideas and concepts that help understanding.⁹²

4.1.2 Difficulties navigating behaviour theories

One of the many difficulties for those who approach the broad subject of behaviour theory in the context of predicting and changing health behaviour is the vast and inconsistent use of new vocabulary. The terminology does not necessarily have inherent or intuitive meaning and words used for constructs from different theories may seem similar or have overlapping meaning without actually being truly interchangeable. Many of the social cognitive models fail to supply clear construct definitions.¹⁶¹ When comparing different social cognitive models there are some constructs, which are very similar (if not identical) to each other but are given a different label. Moreover when definitions of terms are supplied they often use new and equally impenetrable words in the explanation.

In common with all theories of behaviour, the development of these theories is, to use a metaphor, a map of the territory rather than being the territory itself. Each construct is a symbol or landmark on the map rather than the actual thing it purports to represent. Maps are useful to help guide us through a landscape and it may be that a poorly drawn map is better than none. Unless each of the maps uses terminology that is understandable and interchangeable, then the travellers are likely to get confused and lost. However it is important to realise that with every new journey if new discoveries of 'facts' are made the map may need to be modified or otherwise it will become less and less useful. If a new feature (from research or observation) cannot be incorporated into or explained by the map as it stands it may be that the map is the wrong tool for pointing the right direction. In practice where there is a discrepancy between the theory and

observation usually some explanation or addition is offered for the difference rather rejection of the theory. To persist with the metaphor, if a train appears on the landscape, their presence may not be explicable by the ordinance survey map made ten years ago but could be explained by a calendar and the timetable of the local train service (predictable, manmade conforming to some complex pattern), whereas a major flood (catastrophe/unpredictable event) may destroy landmarks and make watery chaos of geography and other features may disappear.

Therefore theories may be able to describe or predict patterns of behaviour but sometimes even a detailed or well established theory may fail due to the excessive number of variables or complexities of the determinants of human behaviour.

4.1.3 What are cognitive factors?

Behaviour may be determined by intrinsic factors and extrinsic factors (see Figure 2, p. 93). Broadly speaking extrinsic factors are external to the individual and consist of incentives (e.g. subsidies) or disincentives (e.g. taxes) and legal structures. Intrinsic factors include individual circumstances, personality and cognitive factors.

The dictionary definition of cognition is 'when you think or use a conscious mental process'. However the psychological definition refers to all processes by which the sensory input is transformed, reduced, elaborated, stored, recovered, and used. Psychologists have concentrated their attention on these cognitive factors as they believe them to be very important determinants of

behaviour and far more likely to be amenable to change. As proximal determinants of change these cognitive factors are assumed to be important causes of behaviour, which mediate the effects of other determinants such as social class. They are also assumed to be more open to change than other factors such as personality.¹⁶¹

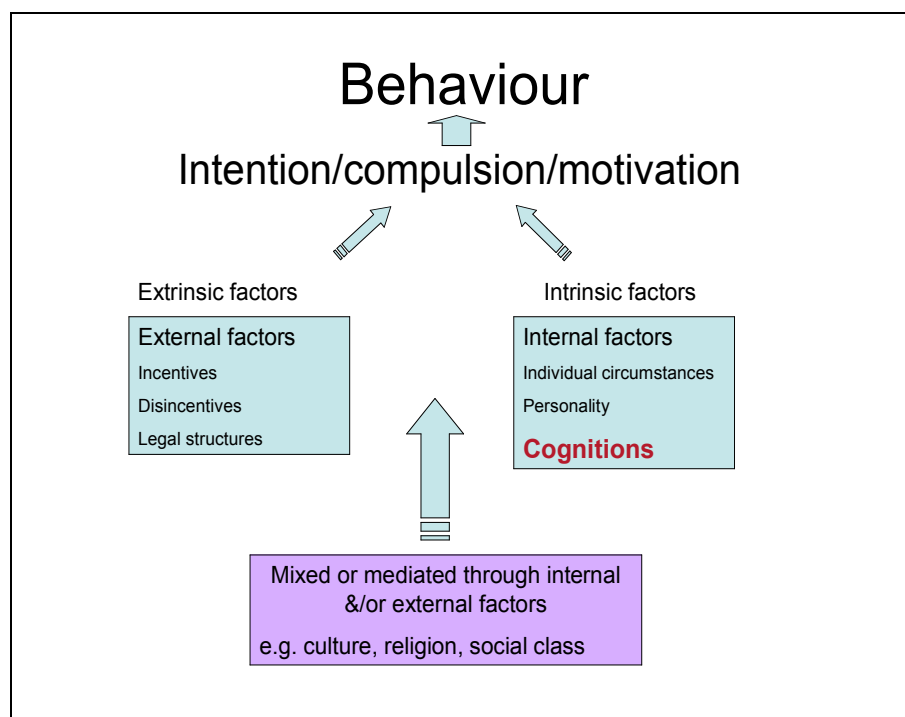


Figure 2 Intrinsic and extrinsic factors in change.

Social cognition models have been developed to describe the processes of how cognitive factors produce behaviour in a social context. They regard cognition as being shared by individuals within the same society.¹⁶² Social cognition models have their origins in the work of Bandura which suggested that behaviour is determined by expectancies, incentives and social cognitions.¹⁶³ The difference between cognition models and social cognition models is that social cognition models include the influence of other people and the society within which the individual is functioning. For example, in

practice this is conceptualised in the construct of 'normative belief': 'My children want me to stop smoking and their opinion is important to me'.

Many models have been developed, some have faded, some have persisted but none have been universally accepted. Theories in psychology often continue to persist unchallenged even when major observations do not fit the theory or there are major discrepancies. Meehl (1978) observed that theories are neither refuted nor corroborated but fade away.¹⁶⁴ When a model is tested in research to predict behaviour or outcomes of an intervention there is usually a gap between those expected and observed outcomes. Indeed a good theory should consist of constructs that are specific enough to be testable and be useful for designing intervention and be able to predict behaviour with reasonable accuracy.¹⁶⁵ The discrepancies between the predicted and observed outcomes are often explained away rather than used to reject or refine the theory.

The gap between prediction (from theory) and observed behaviour demonstrates significant failings of theory to predict outcomes or observations in the real world. Psychologists call this gap 'variance'. This is not the only discrepancy between theory and reality in what I have called the *prediction-intention-behaviour deficit* (see Figure 3, p. 95). Models of health beliefs only predict between 40 and 50% of behavioural intentions.^{166;167} Furthermore, having an intention to behave in a certain way does not reliably predict behaviour. Intentions may explain between 19 and 38% of the variability in behaviour.^{162;162;168}

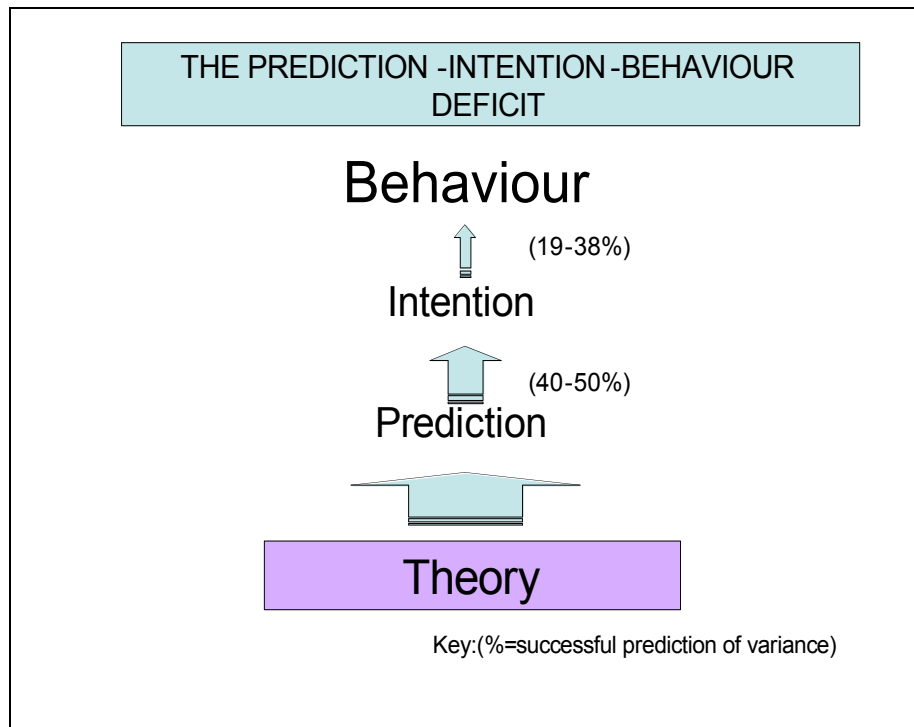


Figure 3 The prediction-intention–behaviour deficit

Thus there is a progressive fall-off in observed (or reported) change from the predictions made by a theory to intention to behave in a certain way and between that and actual behaviour. 'Intention' therefore becomes equivalent to a surrogate measure of outcomes and is not a reliable indicator of actual behaviour

A number of 'get-out clauses' are used in reports of studies that are less than conclusive. For example statements that '*the variables were not operationalised properly*' may disguise either methodological or theoretical deficiencies. Usually they mean that the questions did not test what the researcher hoped they would test. In other studies demonstrating limited success in predicting outcomes the explanation may be 'sample characteristics'. Of course they will only consider explanations that are within the paradigm of their own behaviour theory and

philosophy of knowledge and may therefore overlook other explanations. Further discussion of this possibility is beyond the scope of this PhD thesis.

4.1.4 How are theories tested?

A number of different research designs are commonly used to test these theories. Cognitive theories are mostly tested by cross-sectional design.¹⁶⁹ Each construct within the theory is measured. The most common measure is by questionnaire (but could be by structured face-to-face or telephone interviews) using questions designed to test the extent of a particular construct. After a period of time the outcome behaviour is recorded (e.g. by observation, medical records or self reporting). An attempt is then made to determine to what extent each of the construct variables has predicted the outcome change in behaviour.¹⁶⁹

For example, stage based theories can be tested using cross-sectional surveys where the participants are classified into their stages and compared according to the relevant variables that are theoretical determinants of change between stages.¹⁶¹ Examination of stage sequences and longitudinal prediction of stage transition are used to test whether different variables successfully predict transition from one stage to another.

The strongest evidence for a stage-based theory would be from experimental studies of stage-matched interventions. In other words, behaviour change in one group after an intervention (designed to target a determinant of behaviour change which is theoretically active at one stage rather than another) is compared with another group where the intervention is not matched. Currently a

review has been designed and is being conducted by the Cochrane collaboration to determine if there is any strong evidence for the impact of stage based interventions on smoking.¹⁷⁰

4.2 Theory of planned behaviour (TPB)

This sub-section also includes the related theories of the theory of reason action and behaviour prediction theory)

The theory of planned behaviour is one of a number of social cognition theories. It is an extension of the theory of reasoned action which arose from work by Fishbein and Ajzen who published '*Belief, Attitude, Intention, and Behavior: An Introduction to Theory and Research*'.¹⁷¹ The theory became known as the theory of reasoned action but after several changes was known as the theory of planned behaviour. Changes were made to account for other observed outcomes. The addition of 'perceived control' over the behaviour altered the name to the theory of planned behaviour.¹⁷²

4.2.1 Fundamental structure

According to the theory, behaviour is influenced by the intention to behave in a certain way. Intention is determined by three major variables called subjective norms, attitudes and self-efficacy (also called perceived behavioural control). Self-efficacy had been added to account for times when people have the intention of carrying out a behaviour, but the actual behaviour is 'thwarted because they lack confidence or control over behaviour'.¹⁷³

The theory of planned behaviour suggests that a person's behavioural intention depends on the person's attitude about the behaviour and subjective norms. If a person intends to 'do a behaviour' then it is likely that the person will do it.

Theory of planned behaviour

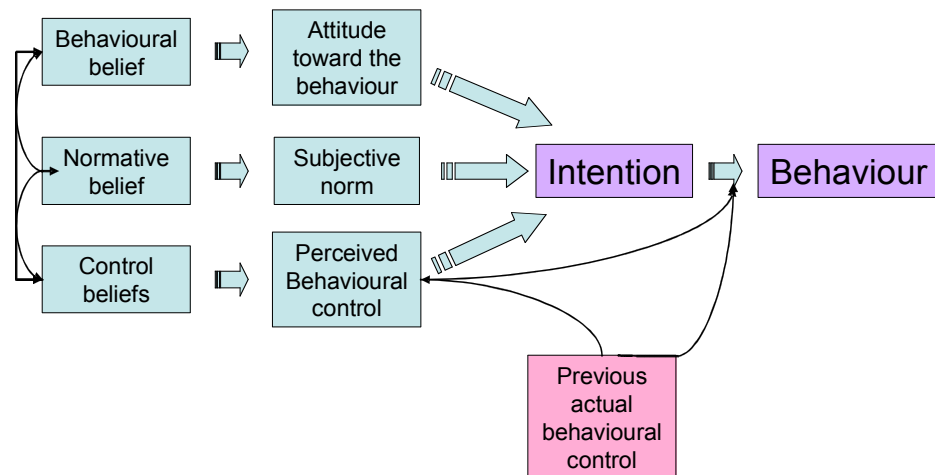


Figure 4 Theory of planned behaviour

Behavioural intention measures a person's relative strength of intention to perform a behaviour. Attitude is comprised of beliefs about the consequences of performing the behaviour multiplied by his or her valuation of these consequences.

Subjective norm is seen as a combination of perceived expectations from relevant individuals or groups along with intentions to comply with these expectations. In other words, 'the person's perception that most people who are important to him or her think he should or should not perform the behaviour in question' (Ajzen and Fishbein, 1975).¹⁷¹ To put the definition into simple terms: a person's voluntary behaviour is predicted by their attitude toward that

Example:

A 50 year old smoker and his wife have been considering their smoking for some time (**motivation to comply**). Their teenage children have always disapproved of their parents' habit (**normative belief**). The nagging has not lessened over time. Mr X is confident he can stop at any time that he wants to; he is in control of his habit and believes that he does it because he enjoys the sociable side of life and fits with his friends (**subjective norms**).

Recently his wife has been getting anxious about the health issues. A friend of Mrs X has told her about her brother who recently had a heart by-pass. She has a cough and gets puffed on the stairs, which she thinks could be due to smoking or just lack of fitness. She is not sure she has the will power to stop easily (**control belief= perceived likelihood of constraining and facilitating conditions**) and her biggest worry is putting on more weight. She already struggles with her weight (**constraining condition**) and her friend put on 2 stone when she stopped last year. The friend's husband believes it is better to be slim and look good than to be a fat non-smoker (**subjective norms**). However she thinks that if there are no fags in the house and her husband stops too (**facilitating condition**) – maybe she can succeed (**control belief x perceived power= perceived behavioural control**)

Both Mr X and Mrs X intend to stop (**behavioural intention**) smoking but will this translate into action and change of behaviour?

Motivation to comply x normative belief = subjective norm

Control belief x perceived power=perceived behaviour control (efficacy)

Behavioural belief x evaluation of behavioural belief = attitude

Box 2 Example of TPB and smoking.

behaviour and how they think other people would view them if they performed the behaviour. A person's attitude, combined with subjective norms, forms their behavioural intention. However, Fishbein and Ajzen state that attitudes and norms are not weighted equally in predicting behaviour. 'Indeed, depending on the individual and the situation, these factors might have very different effects on behavioural intention; thus a weight is associated with each of these factors in the predictive formula of the theory. For example, you might be the kind of person who cares little for what others think. If this is the case, the subjective norms would carry little weight in predicting your behavior'.¹⁷³ Miller (2005) defines each of the three components of the theory as follows.¹⁷³ (I have used different examples to illustrate aspects of the theory):

Attitudes:

Attitudes are the accumulation of beliefs about a particular behaviour weighted by evaluations of these beliefs. Someone may believe that exercise is good for health, that exercise improves looks, but that exercise takes too much time, and that exercise is uncomfortable. Each of these beliefs can be weighted (e.g., health issues or looks might be more important to an individual than issues of time or comfort).

Subjective norms:

Subjective norms refers to the influence of people in their social environment on their behavioural intentions; different opinions or beliefs of different people will influence a person's behavioural intention to varying degrees. In the example (Box 2, p. 100) Mrs X's children have some influence but the belief of a friend about weight gain (after smoking cessation) and another about health risks will

be weighed against each other to determine behavioural intention which will lead to stopping or failure to stop smoking.

Behavioural intention:

Behavioural intention is a function of both attitudes toward a behaviour and subjective norms toward that behaviour, which has been found to predict actual behaviour.

4.2.2 Research background

The theory of planned behaviour continues to be used very widely in many different cultural contexts in health and other psychological research to predict and explain a diverse range of behaviours, including health-relevant behaviours such as smoking,¹⁷⁴ condom use, exercise and diet as well as non health issues such as use of the internet and accessing services or breaking driving rules.¹⁷⁵⁻¹⁸⁰ Many prediction studies are aimed towards identifying beliefs that could subsequently be targeted by a persuasive message.

The process of developing a prediction study typically follows three steps.

Firstly, the most frequent important beliefs are elicited from a sample of the target group. Secondly, a questionnaire is constructed to assess beliefs that distinguish intenders from non-intenders, and to identify the relative contribution of the attitudinal or normative component. Finally, an intervention is designed to change the key beliefs identified.

Ogden reports a search of four major psychology journals for articles published over the previous five year period, which purported to test or apply one or more

social cognition theories.¹⁶⁵ She concluded that the majority of the articles did not strongly support the models being used. In particular there was little evidence for the expected associations between variables or for the models' ability to predict the particular outcome being studied. All of the articles examined left much of the variance unexplained, with explained variance ranging from 1% to 65% for behavior and 14% to 92% for behavioral intentions. But, even though the variables were not predictive and the variance explained was low, the research data were not used to dismiss or disprove the validity or reliability of the particular models tested. This emphasizes the general criticism of behavioural models that they are not disproved or rejected but are simply allowed to fade.

When constructs are not defined or are too vague, precise testing is impossible, and a theory cannot be 'disproved'. In practice this means that if one out of the three main variables (subjective norms, perceived behavioural control, attitudes) does not appear to have any predictive value, several explanations are offered but no data are collected to demonstrate that the model is wrong. Therefore the theoretical basis remains unchallenged.¹⁶⁵

However, in response to these objections, Azjen argues that 'there is nothing in the theory (of planned behaviour) to suggest that attitude, subjective norm and perceived behavioural control will each make a significant contribution to the prediction of intention. The relative importance of these three factors is likely to vary from one behaviour to another and between populations'.¹⁸¹ This highlights the importance of precise definitions and ability to test precisely and reliably. In

situations where the theory has failed to accurately predict behaviour the investigator may give explanations while failing to reject the theory ¹⁸² or be tempted to add a variable such as 'habit', 'ambivalence', moral obligation and self efficacy.¹⁸³

In a thorough systematic review of the application of the theory of planned behaviour in interventions for behaviour change Hardeman et al found 30 papers describing 24 distinct interventions.¹⁸⁴ Methods of change generally involved persuasion, information, skills development and goal setting. In one half of the studies the theory was used to develop the intervention and in the remainder the theory was used to assess changes. Approximately half of those studies involving an intervention reported a change of intention but the effect size was small. TPB was mostly used to measure the process and outcomes rather than to develop the intervention but studies were rarely explicit about the use of TPB.

4.2.3 Critique of the theory of planned behaviour

Sheppard et al. suggest 'that more than half of the research to date that has used the model has investigated activities for which the model was not originally intended'.¹⁸⁵ In other words the model is frequently applied to target behaviour and situations where the subject does not have voluntary control, choice is not a reality or intentions are assessed where it is impossible for all the necessary information to be available. Many of the studies in their meta-analysis involved goals and choosing alternatives. Goals were judged by the presence of significant barriers to their fulfillment e.g. resources, training or the cooperation of others. Their expectation was that the model would not fare well in such

situations. However, they found the model 'performed extremely well in the prediction of goals and in the prediction of activities involving an explicit choice among alternatives'. Thus they concluded that the model 'has strong predictive utility, even when utilized to investigate situations and activities that do not fall within the boundary conditions originally specified for the model. That is not to say, however, that further modifications and refinements are unnecessary, especially when the model is extended to goal and choice domains'.¹⁸⁵

Hale et al. also echo these views when they state 'The aim of the TPB is to explain volitional behaviours. Its explanatory scope excludes a wide range of behaviours such as those that are spontaneous, impulsive, habitual, the result of cravings, or simply scripted or mindless. Such behaviours are excluded because their performance might not be voluntary or because engaging in the behaviours might not involve a conscious decision on the part of the actor'.¹⁸⁶

Sheppard et al. (1988)¹⁸⁵ are supportive of the theory of planned behaviour but make exceptions for certain situations when they say 'a behavioural intention measure will predict the performance of any voluntary act, unless intent changes prior to performance or unless the intention measure does not correspond to the behavioural criterion in terms of action, target, context, time-frame and/or specificity'. So, for example, if a smoker learns they have a medical condition made worse by putting on weight (e.g. back pain), and they believe they will put on weight after stopping smoking, this may affect their behavioural intention (about quitting).

Sheppard et al. (1988) propose that there are three limiting conditions on the use of attitudes and subjective norms to predict intentions and intentions to predict the performance of behaviour:

1. Goals versus behaviours:

There is a distinction between a goal intention (cutting smoking from 30 to 20 per day) and a behavioural intention (taking nicotine replacement therapy).

2. The choice among alternatives:

The presence of choice may dramatically change the nature of the intention formation process and the role of intentions in the performance of behaviour.

3. Intentions versus estimates:

There are occasions when what a person intends to do and what they expect to achieve are different.

4.2.4 Smoking cessation and TPB

Two basic assumptions cast doubt on the validity of TPB in the context of smoking cessation. The first assumption is that (undesirable) behaviour is under voluntary control. However much mankind believes that he is in control, the evidence from clinical and practical life-experience demonstrates that self-control is difficult and inconsistent. Even without chemical dependence the behaviour under investigation may be habitual or have deep psychological roots in a person's life journey or as a personality trait.

The second assumption is that people are rational. If that were so then open access to facts about the consequences of certain behaviour would translate into safer conduct in all spheres of life. However people are capable of dismissing information or weighing up the pleasurable effects of the 'here and

now' which may undermine any logical conclusions. People may take extraordinary risks with personal health or safety, for short-term gains or experiences (see Section 3.2.2, Addiction, p. 50).

TPB has been used as the theoretical basis of a number of smoking research studies.^{174;187;188} Coleman et al. used questionnaires to collect data on the intentions of about a 1000 smokers attending general practice. Using the theoretical framework of TPB they concluded that where smokers perceived that their problems were smoking related they were more likely to have tried stopping in the past (odds ratio 1.78, 95% CI 1.26-2.67), to want to stop smoking (OR 1.83, CI 1.15-2.9) or to intend to stop in the near future.¹⁸⁸ In other words, in the language of TPB, behavioural intention is formulated by beliefs about health and the attribution to the behaviour. The study did not report any plan to follow up the smokers to confirm whether or not the intentions were translated into successful behaviour change.

The study by Black et al. was a telephone survey of the attitudes and control beliefs of nearly 200 American university campus students who smoked.¹⁸⁷ Only two components of TPB (attitudes and control belief) were measured with questions designed to assess their interest in involvement in different types of smoking cessation programmes. There was no actual intervention and duration of follow up and change in intention or behaviour was not reported. Therefore, it is difficult to make any firm conclusions about how useful the model is the context of smoking behaviour.

There is now plenty of evidence that many decisions about change in behaviour are not necessarily planned. The trigger may be sudden and unexpected and the change equally 'out of the blue'.¹⁸⁹ In the case of smoking cessation West et al found that in their sample half of people who tried to quit did so without any pre-planning. A greater proportion of them were still non-smokers after six months than those who had planned a quit attempt. These results are used to argue that the trans-theoretical model is also deficient.

Existing theories do not predict behaviour particularly well. Some unpredictability will remain even if the theories are improved. TPB continues to be used widely in consumer and health research and has the potential to underpin smoking cessation strategies with appropriate modifications where choice and resources and goals are considered. The theory accounts for changes in intention and a minority of research has clarified the usefulness of the theory in predicting successful behaviour change.

4.3 Health belief model (HBM)

The health belief model (HBM) was developed by Rosenstock in the 1950s for studying and promoting the uptake of services offered by social psychologists and in particular health education programmes. Subsequent amendments to the model were made as late as 1988, to accommodate evolving evidence generated within the health community about the role that knowledge and perceptions play in personal responsibility. It was recognised that effective health education was needed to target potentially modifiable characteristics which determine behaviour.

The relationship between health beliefs and behaviour was conceptualised in terms of 'valence'. Valence is the concept of making behaviour more or less attractive. Derived from value expectancy theory, the HBM has been widely applied to many different preventive health behaviours as well as actions taken to seek medical attention for illnesses.¹⁹⁰

4.3.1 Fundamental structure of health belief model

The original model postulated that preventive health behaviours may be predicted by the following key beliefs that shape health behaviour:

1. The likelihood of experiencing a health problem (e.g. perceived susceptibility to tuberculosis)
2. The severity of the consequences of that problem (tuberculosis)
3. The perceived benefits of particular health behaviour (belief that TB could be asymptomatic and screening could detect it early) (see Figure 5, p. 110)

The factors influencing behaviour may be divided into threat perception and behavioural evaluation. As above the threat perception is about how much the individual thinks they will get the condition and the consequences of getting it. Behavioural evaluation distinguishes between the benefits of change (efficacy) and the personal cost which may be physical, psychological or financial (barriers).¹⁹¹ There is no formula for weighing the balance between cost and benefit (belief) and therefore the construct includes these as separate (independent) variables.¹⁶⁹

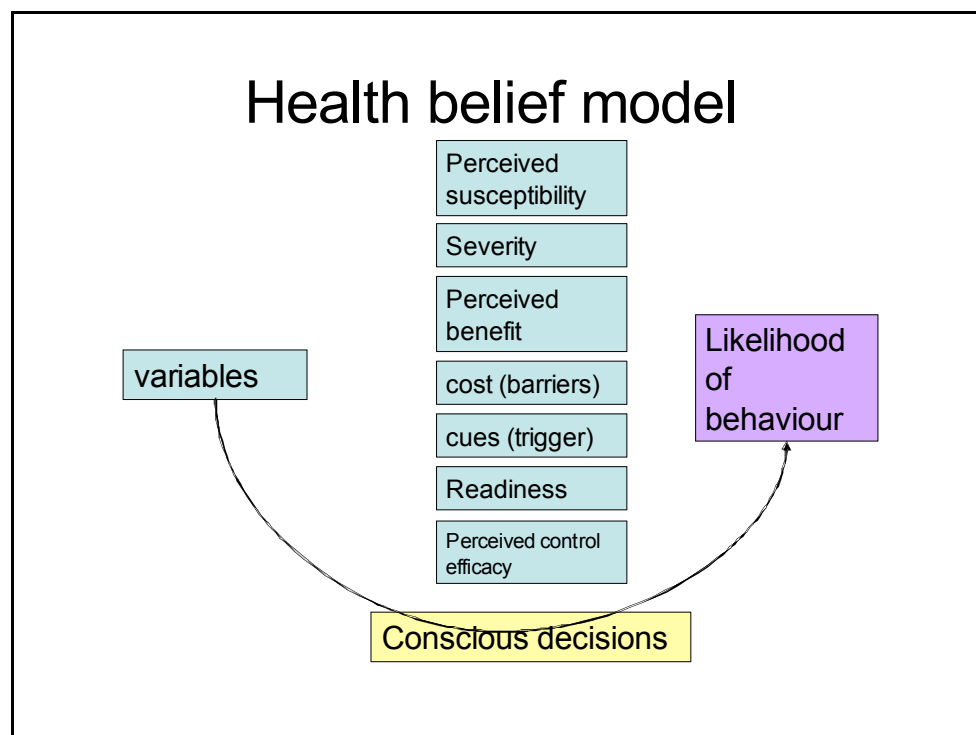


Figure 5 Health belief model

Even when an individual believes in a health behaviour there still may need to be a cue or a trigger to activate the behaviour. Later additions to the model include readiness (to be concerned about health matters) and perceived control over the behaviour (see Figure 5, p. 110).

4.3.2 Research background

The initial prospective research demonstrated correlation between three factors or constructs:

- likelihood of experiencing a health problem
- the severity of the consequences of that problem and
- the perceived benefits of a health behaviour

and the uptake of the health behaviour (screening) and therefore were believed to be predictors of behaviour.

Further research demonstrated that a health education intervention designed to increase perception of susceptibility, severity and anticipated benefits, increased health improving behaviour.¹⁹²⁻¹⁹⁴ A meta-analysis of studies using this model in adults highlighted some of the problems with definitions of the construct variables.¹⁹¹ Interestingly they identified over 200 published studies for analysis but only 16 fulfilled their criteria for inclusion by measuring all the major components of the theory (now increased to four- susceptibility, severity, benefit and barriers). The authors concluded that 'the weak effect sizes and lack of homogeneity indicate that it is premature to draw conclusions about the predictive validity of the HBM as operationalised in these studies'.

Two constructs were later added. Perceived efficacy which is an individual's self-assessment of ability to successfully adopt the desired behaviour and cues to action which are external influences promoting or triggering the desired behaviour.¹⁹⁵ The second addition was cues to action which can include internal cues such as perception of body states and external cues such as interpersonal

interaction and mass media communication.¹⁹¹

As with the other models already described the HBM has been used in a wide variety of settings including the study of uptake of healthy related behaviour by uptake of tuberculosis screening, vaccinations, cervical cancer screening and preventative dentistry.¹⁶⁹

Some studies have given support to the model especially when applied to individual components. In other words if each construct is examined in isolation they are shown to be predictors of health behaviour in selected areas of study. This is especially so in the area of health screening where 'barriers' and 'perceived susceptibility' were found to be the best predictors of healthy behaviour. For example, knowledge about breast cancer is correlated with breast self-examination and having regular mammograms.¹⁶²

4.3.3 Critique of the health belief model (HBM)

There is a difference between demonstrating that individual components of HBM predict behaviour and showing that the combination of constructs is a useful predictor of behaviour. For the HBM to be validated as an integrated model of behaviour each of the dimensions has to fit with each other and the research needs to demonstrate the relative importance of each as well as their interactions with one another. In other words the theory must be demonstrated in practice and in research.¹⁹¹ As discussed above, reviews of research studies using HBM show only weak correlations and their heterogeneity mean that it is difficult to draw conclusions about the predictive value of the HBM. Few studies meet minimal criteria for valid representation of the HBM components indicating

that future studies should focus more on systematic analysis of the constructs of HBM.¹⁹¹

Conflicting findings are found in some studies which confuse matters.^{189;190} For example it would be reasonable to predict that *high susceptibility* would be correlated with health seeking behaviour whereas some studies found the opposite.¹⁶² Other studies showed that *benefits* of carrying out a health behaviour (stop smoking, save money) and perceived severity of the consequences (lung disease) were not related. Furthermore, the model focuses on conscious decision making. It makes no allowances for other external forces such as social pressure or perceived controls or influences from other people and finally, there is no clear indication of the relationships between the components.

4.3.4 Smoking and the health belief model

The HBM has been used to develop programmes or to predict outcomes in a number of studies about smoking cessation interventions.^{97;196-202} Previously, the dubious quality of studies has been a real problem in determining the usefulness in smoking cessation. The meta-analysis by Harrison et al in 1992 not only found few studies which met their strict quality criteria but also failed to identify any well conducted studies that related to smoking behaviour.¹⁹¹

Few studies using HBM have been of sufficiently clear design to separate out the influence of the different components or have been criticised for not fully operationalising the variables.²⁰³⁻²⁰⁷ One study combined the two constructs *benefits* and *barriers* into a single 'index'.¹⁶⁹ The prospective study by Mullen et

al was a comparison of three different health models in four different health areas (smoking, exercise and consumption of fried and sweet foods).²⁰⁸ As far as smoking is concerned they confirmed that the construct *susceptibility* to serious illness over the next ten years was associated with an attempt to quit smoking and a reduction of cigarettes smoked. They concluded that in such areas as smoking, where health risks are widely known, personalization of consequences may be important in influencing behaviour.²⁰⁸ The construct *barriers* was operationalised as 'attachment to aesthetic pleasures of smoking'. This did not directly predict change in behaviour in this study.

Those with pre-existing morbidity, either caused by smoking or made worse by smoking, are a special risk group. A prospective questionnaire survey of nearly 400 patients attending a clinic for respiratory diseases measured health beliefs, smoking history and patient predictions of future smoking behaviour.²⁰⁵ Quit rates were low and predictions had low accuracy. Very few firm conclusions about the constructs of HBM could be drawn but the study confirmed the experience and perceptions of clinicians that smokers with lung disease represent a group resistant to change.

A small retrospective study of diabetics comparing the variables of the HBM among smokers and ex-smokers concluded that perceptions of severity, perceptions of susceptibility, cues to action, and the modifying factor social support were useful predictors.²⁰⁶ However, they point out that recall of physician advice may not be reliable, as it seems unlikely that less than half of the diabetic patients were advised to stop smoking. The most likely explanation

is a combination of failure of retrospective data and memory and resistance or denial. Those with chronic diseases made worse by smoking are resistant to change and may build up a strong resistance to professional advice.

In conclusion, despite having intuitive appeal and face validity, the HBM model has been shown empirically to be a poor predictor in the context of smoking behaviour and cessation.

4.4 Trans-theoretical model (TTM)

Among the stage theories of behaviour change the trans-theoretical model is probably the best known and most widely used in addiction and smoking research. It is beyond the scope of this thesis to discuss all the stage theories but I will put the trans-theoretical model into context of stage theories in general.

At the simplest level any stage theory would have two discrete stages with the ability to move from the first to the second under the influence of independent variables. The transitions are the dependent variables. The subject either remains the same or moves (dichotomous stages). A three-stage model would have three separate states or stages with independent variables between each stage. The independent variables may act alone or via others at different stages. Complexity may increase with the numbers of stages and the ability to move in either direction and to skip stages.

The trans-theoretical model (TTM) was originally developed by psychologists Prochaska and Diclemente over 30 years ago and has been developed and used widely in researching the process of change.²⁰⁹⁻²¹¹ The model has been applied to a wide range of behaviours including smoking, drinking, drug use, uptake of mammography screening, and healthy activities (exercise, healthy eating, safe sex).

The name 'trans-theoretical' arises from the fact that the model incorporates 15

theoretical constructs and was an attempt to draw these into a single framework.²¹² It has been modified several times since its original development.

4.4.1 Fundamentals of trans-theoretical model

The exponents of this model describe five stages (Box 3 and Figure 6, p.120).

1. Pre-contemplation: not intending to make any changes
2. Contemplation: considering a change.
3. Preparation: making a small change.
4. Action: actively engaging in a new behaviour.
5. Maintenance: sustaining the change over time.

Box 3 General meaning of stages

The pre-contemplative stage corresponds to that period where there is no intention to change in the foreseeable future and the person has no faith in their ability to change and suppresses any thoughts about change. They may or may not have sufficient information about the risks of their behaviour but avoid engaging in dialogue about their habit and its effects. There is a tendency to overestimate the benefits of the behaviour (e.g. pleasure of smoking) and underestimate the hazards. It is unlikely that this group will volunteer, or respond to an invitation, to engage with smoking cessation treatments or educational programmes to discover more about risks to their health. They may regard change as pointless as they may doubt their ability to change. Researchers often exclude this category from intervention studies.

Commentators of this model have identified different aspects of the pre-contemplation stage. This has been termed 'resistance and the four Rs'. Reluctant pre-contemplators are those who through lack of knowledge or inertia do not want to consider change. Knowledge and empathy are most helpful for them.¹⁴² There is reluctance rather than resistance and is this not particularly conscious. Rebellious pre-contemplators have a heavy investment in the behaviour and resist any attempt to being told what to do. They will be hostile and resistant to change. If they are recruited to any programme they will insist that they are not going to change and make it clear that they will energetically resist. Resigned pre-contemplators do not have that energy and do not feel in control. They have given up the possibility of successful change and feel overwhelmed. They often have a track record of multiple attempts with different techniques. The rationalising pre-contemplator has all the answers, and counter arguments to personal risk and is not considering change.¹⁴²

Contemplation equates with a stage of serious thought about changing within the foreseeable future (often said to be six months). The pros and cons of the habit become fairly equal although there is still doubt about the benefit and continuing to think about change may be a substitute for action. This stage may become prolonged.

The preparation stage is said to exist when there is an intention to change in the very near future (often within the next month). They usually have tried to change

over the previous twelve months and are making moves to delay and reduce the harmful behaviour. They already regard the hazards the behaviour as greater than the benefits.

The final two stages are action and maintenance of the new behaviour. Unfortunately people are most likely to relapse in the action stage but as people move towards maintenance of the behaviour (e.g. not smoking) they will increasingly use strategies to avoid high risk situations by working on a healthier lifestyle and modifying their environment to reduce exposure to temptation.^{210;213}

4.4.2 Constructs of the trans-theoretical model

The trans-theoretical model of stages of change has been further developed using a number of different constructs. Instead of the point prevalence (e.g. of non-smoking) being used to measure success of an intervention, Velicer et al.²¹³ propose a three-construct outcome model (habit strength, positive evaluation strength, and negative evaluation strength), where different constructs are sensitive to change for different aspects of the change process over time. This is a complex model which looks at the presence or absence of certain psychological and environmental or physical factors and how each one will influence an individual depending on their stage in the trans-theoretical model (pre-contemplative, contemplative etc). A simple way of looking at their construct is to say that how a person is influenced by any given internal or external factor is determined by their stage of change in behaviour (see Figure 6, p. 120).

Habit strength

For example 'habit strength' is one of the three constructs. A pre-contemplator will be immune to environmental influences. However, recent introduction of laws to ban smoking in certain places may determine where they go but not their smoking status. It is not clear whether this sort of legislation helps or hinders change for pre-contemplators as they may simply become more entrenched in their own belief in a right to choose their lifestyle and habits. A contemplator or action stage person will be very influenced by environment and will attempt to avoid certain situations in order to maintain resolve. They are however vulnerable to situations where they are not in control and may relapse. The maintenance phase is much less sensitive. These ideas are mostly intuitive.

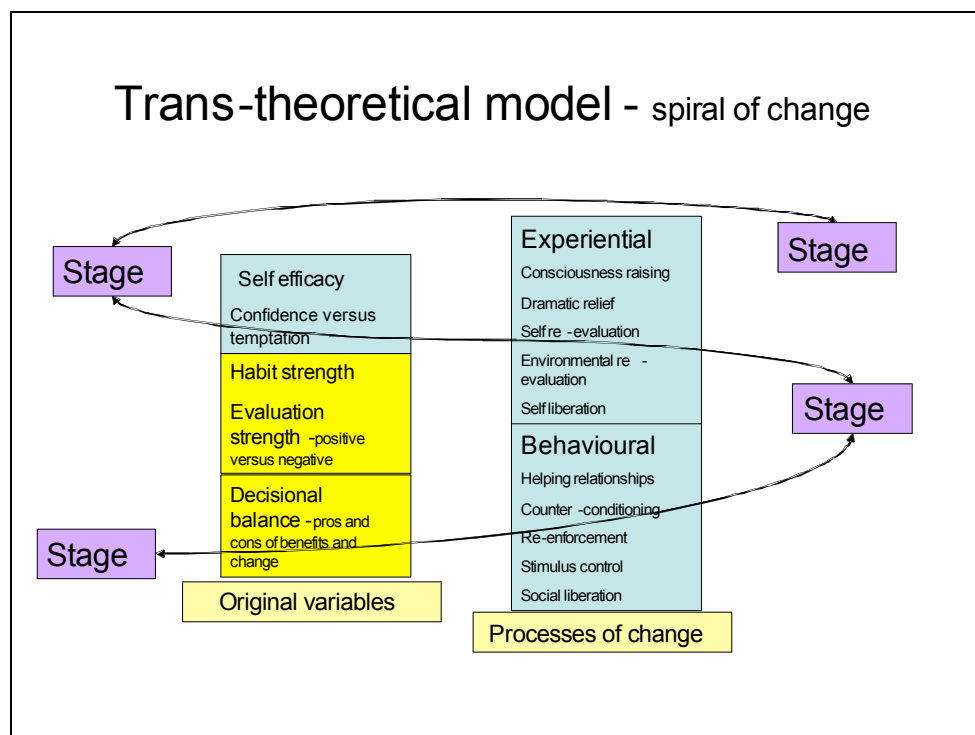


Figure 6 Trans-theoretical model spiral of change.

Evaluation strength

However the other two constructs are less easy to understand. The idea of 'positive evaluation strength' is concerned with belief about the behaviour and how the individual's belief changes or is influenced by information. This construct demonstrates a positive attitude to the benefits of change until what the authors have called a 'conviction threshold' is met and therein lies the opportunity for change. Dipping through the conviction threshold into a position where information is received and processed conceptualises a teachable moment in an otherwise resistant individual. In contrast the most sensitive time for processing information is during the action stage. Negative evaluation strength is in a sense the reverse image of the positive evaluation strength. The important difference is that when contemplating negative aspects of smoking the individual may ignore it because of indifference or from lack of conviction. Pre-contemplators and people in maintenance are both indifferent whereas those in the contemplation phase are still not convicted fully of the negative influences of their smoking but if and when they do move into the Action stage they are not indifferent and start becoming convicted and are open to processing information that will help them on their journey to cessation.

Other constructs

Prochaska et al have introduced other constructs.²¹⁴ Self-efficacy is said to be a balance of confidence and temptation. That is, the confidence that one can engage in the healthy activity or the temptation to engage in the unhealthy activity across different challenging situations. Decisional balance is divided into the pros and cons of benefits and change. It is unclear how the original

constructs (Figure 6, yellow box) (p. 120) and the more recent constructs and processes of change (Figure 6, blue boxes) are related or whether the more recent work supersedes the former.

They have identified ten processes of change (Figure 6, blue boxes), subdivided into the experiential processes (such as consciousness raising, dramatic relief, self re-evaluation, environmental re-evaluation and self liberation) and behavioural processes. Consciousness-raising encompasses the influence of new facts and ideas that support the healthy behaviour change. Dramatic relief is a term, which paradoxically indicates the unpleasant negative emotions from the perception of risk from the unhealthy behaviour. Self re-evaluation is about the realisation that ones identity includes behaviour (change), whereas environmental re-evaluation is about the negative or positive impact on ones social or physical environment of behaviour. Self-liberation is a positive commitment to change.

Behavioural processes are more concerned with social, external interactions. They are categorised into helping relationships, counter-conditioning, reinforcement management, stimulus control and social liberation. For example these include the following; seeking and using social support (helping relationships), substituting healthy alternatives (counter conditioning) or rewards (reinforcement management) for the healthy behaviour, removing reminders and decreasing rewards (reinforcement management) for unhealthy behaviour (stimulus control). Social liberation is the realisation that social norms are changing towards support for the healthy behaviour change.

In summary, the stages of change may be represented as a dynamic spiral rather than simply as linear progression from one stage to another in orderly fashion.¹⁶¹ People start at the bottom of the spiral, in pre-contemplation. They then move through the stages (contemplation, preparation, action, and then maintenance) but will typically relapse back into an earlier stage. They may cycle and recycle through the stages several times before reaching the top of the spiral and achieving successful long-term behaviour change. On a practical level the authors claim that people can be categorised into the stages by the use of three simple questions of intention.

4.4.3 Critique of the trans-theoretical model

The usefulness of this model has been seriously challenged in recent years by a number of authors.^{161;165;215} The biggest objection to this model is the arbitrary division into stages based on a time of intention. The stages are assumed to be discrete whereas in reality they usually represent a continuum of personal experience and behaviour. The theory should allow for a jump between non-adjacent groups but the assumption is that one moves through or spirals round the stages.

At first glance this model may have a logical appeal and seem theoretically plausible but practical experience and research has shown big changes in behaviour may occur without a stage shift. When defined, by intention and attempts at change, the groups would appear to be homogenous. In reality this is far from true. As I have explained the concept of 'stage' is not as simple as it

first appears as each level includes numerous variables. If a smoker declares that they intend to stop smoking in the next four weeks an external observer might reasonably expect that they are likely to be more successful at cessation than the person who says that they are not considering any change. However, West et al have found that nearly 50% of quit attempts happen in an unplanned way and that these unplanned event have better long term success than those which are planned.¹⁸⁹ Furthermore there is little consistent evidence of progression between stages to support the theory.²¹⁶ At its simplest the five-stage model (see Box 3, p. 117) could be replaced with a simple question 'are you planning to change?' A continuum rather than distinct states is more likely.

A further objection to this model is that the creators have not established a clear cause and effect relationships between different variables. Studies have been done using cross-sectional surveys to determine stages and compare the presence of relevant variables that are theoretically active determinants of change between stages. It is still unclear whether or not the stages are truly 'predictive' of behaviour and whether or not interventions can be targeted at a given stage.²¹³

Cross-sectional surveys do not allow conclusions to be drawn about the individual role of presumed causative variables. Sutton reviewed the research designs that have been used to test predictions from stage models (cross-sectional comparisons of people in different stages; examination of stage sequences; longitudinal prediction of stage transitions; and experimental studies of matched and mismatched interventions) and concluded that evidence for the

model is inconsistent.²¹² Experimental and longitudinal designs would give more information but are rarely done.

So-called 'stage-based' interventions would be very useful in a treatment setting if they are valid.²¹⁶ If different variables are active and causative at a certain 'stage' then treatments or interventions would be more effective if targeted at the correct stage. There would be a great benefit if interventions could be targeted towards particular groups based on psychological factors, which would give cost effective interventions. In theory this would save time and money by avoiding using methods that are ineffective for certain groups and maximising positive change, and perhaps minimising potential harm by using inappropriate methods in the 'wrong' contexts. However, so far evidence is lacking for this approach

Finally the model focuses on conscious decision-making and assumes that people make clear plans, which they are able to act upon. Although we like to believe that we are rational decisive beings the evidence is contrary to this notion. Crises, sudden changes of mind and life events may cause sudden shifts and spur of the moment changes.¹⁸⁹

4.4.4 Smoking and the trans-theoretical model

For non-psychologists, the simplicity of application of the TTM in classifying the 'stage of change' of smokers is probably the greatest attraction of this model. Many researchers have used this model as another measure of baseline characteristics of participants rather than as a means of testing the model for its value at predicting behaviour change.^{56;155;159;217;218} However, very little

prospective randomised research has used the model to test stage based targeted interventions.

Those with chronic diseases attributable to smoking can be classified into the stages of the TTM. In some studies a particular stage has been correlated with how susceptible they are to change in behaviour. In a study of those with established heart disease people who were classified to the preparation stage were four times as likely to have quit six months later than those who were in the contemplative stage. There was even a doubling of quit rate of those starting in the contemplative stage compared to the pre-contemplative.²¹¹

The idea of stage of change can also be conceptualised in the theoretical framework proposed by Glass et al –the nested hierarchy of systems which allows for a theoretical heightened sensitivity to exposure.²¹⁹ Other researchers have explored the possibility of a ‘window of opportunity’ such as during hospital admission.^{123;220} If attempts are made to induce change in behaviour at the wrong time or context the result may be confrontation and failure.¹⁴² This may go some way to explain the conclusion of the Cochrane review that community based interventions have not shown any reduction in smoking or indeed may have negative affects.¹¹⁶

As far as stage based interventions and smoking are concerned van Sluijs et al, conducted a systematic review of stage based controlled trials which had been initiated in primary care with the purpose of behaviour change in three specific

areas- smoking, physical activity and diet.²²¹ Using an explicit ten point scoring system they judged methodological quality of each of the studies. Among the studies judged to be of sufficient methodological quality to analyse in detail, fourteen of the studies were aimed at smokers. They excluded studies that combined behavioural and pharmacological interventions such as NRT. All the interventions were aimed at altering behaviour.²²² Their conclusions were not encouraging. The results for smoking were at best indicating a 'positive trend' but without strong evidence for stage based interventions. The trends were shifts in one stage or increased quit attempts without conclusive evidence in significant behaviour change in the long term.

Despite the reservations and limitations of this theory the TTM may still remain a useful and very practical framework. Until there is stronger evidence against the TTM and a better and more user-friendly alternative, I believe many researchers are likely to continue to use it at least as a comparative baseline measure in controlled trials and possibly as the theoretical basis of other intervention trials.

4.5 Catastrophe, chaos and complexity theory (CCC)

‘What we call chaos is just patterns we haven’t recognized yet’.

Chuck Palahniuk^g

4.5.1 Basic concepts of catastrophe, chaos and complexity theory^h

Chaos theory is a branch of mathematics that deals with the way in which tensions develop in systems so that even small triggers can lead to sudden changes. During the past two decades, a mixed group of physical, social, biological, and computer scientists have devoted increasing attention to two related disciplines: chaos theory and complexity theory. Often termed non-linear dynamics because they seek to understand systems that change in ways that are not amenable to the linear cause and effect models these theoretical perspectives are thought to have application across a wide range of scientific and social scientific disciplines.²²³

Much of the study of change in health behaviour has been based on the cognitive-rational paradigm in which change is conceptualised as a linear, deterministic process. Determinism is understood as the proposition that a chain of events or rational thought determines everything, including human cognition, behaviour, decision and action. Cognitive behaviour models mostly assume a linear process of individuals deciding about change based on

^g 20th Century American writer- http://en.wikipedia.org/wiki/Chuck_Palahniuk

^h CCC is used to refer to catastrophe &/or chaos &/or complexity theory –some purists would not agree that they are the same but for the purpose of this thesis they are regarded as such as the medical literature often uses the terms interchangeably.

judgments of benefits and disadvantages of their actions. Such a view, fails to account for non-linear, quantum influences on human thought and action. According to the CCC (relevant to understanding health behaviour-change):

- 1) Chaotic systems can be mathematically modelled but are nearly impossible to predict;
- 2) Chaotic systems are sensitive to initial conditions;
- 3) Complex systems involve multiple component parts that interact in a non-linear fashion; and
- 4) The results of complex systems are often greater than the sum of their parts. Therefore, small changes in knowledge, attitude or efficacy may dramatically alter motivation and behavioural outcomes.²²⁴

Advantages of this model over cognitive behaviour models are found in the frustration encountered in trying to explain variance as illustrated and discussed in Section 4.1.3 (see Figure 3, p.95) and Section 4.6 (p. 134). Those linear models resort to dismissing or explaining such problems as errors. The chaotic view allows for the unpredicted and unpredictable aspects seen in human behaviour. However, the linear and chaotic paradigms are not mutually exclusive, as behaviour change may include both chaotic and cognitive processes. Studies of addiction suggest that many decisions to change are sudden and unplanned (so called 'quantum'). Any model, which hopes to endure and gain credibility, must include both aspects of observation of human behaviour change.

The trigger for change may also be conceptualised as a 'tipping point'. This idea

was popularised in *The Tipping Point: How Little Things Can Make a Big Difference* by Malcolm Gladwell.²²⁵ Tipping points are dramatic changes in social behaviour that arise quickly and usually unexpectedly. These come in a variety of guises, including advertising slogans or tunes, political ideas or shocks in the stock market, and most are impossible to predict. However, retrospectively analysis may produce a variety of possible rational explanations for the phenomena. In a similar way, smoking may stop or restart in an unpredictable manner with some plausible explanation when viewed with the benefit of hindsight. In the same way as these phenomena may be unpredictable but explicable in hindsight, some interventions may work through mechanisms that cause a shift in behaviour in a particular direction.

4.5.2 Research and biomedical application

CCC has been applied to the study of health related behaviour for at least 40 or 50 years. However, compared to many other theories within the field of psychological and biomedical research there are far fewer references to this model.

A database search of the literature using the search terms catastrophe or chaos or complexity theory and their use in biomedical or psychological research, delivers a small group of papers. Within this group are a wide variety but limited number of studies including research of; relapse in alcoholics²²⁶; progression of leprosy²²⁷; clinical course of patients in an intensive care unit²²⁸ and analysis of sudden unplanned smoking cessation.¹⁸⁹

The CCC model was found to predict the largest proportion of the variance in

both alcohol and marijuana use among adolescents. Results suggest that a CCC model of adolescent behaviour can provide an important and new way of conceptualising risky behaviour.²²⁹ However, their analysis of smoking interventions for adolescents did not find the model very useful compared to interventions for other types of risky behaviour.²³⁰ Recently this idea has been revisited by a number of researchers who have become dissatisfied with the other established and overused theories (in particular TTM) and are looking for a better way of predicting or explaining behaviour change in smoking and other addictive behaviours.²³¹

4.5.3 Critique of catastrophe, chaos and complexity theory

The practical usefulness of any theory is in its ability to predict outcomes of psychological interventions and then to be able to design interventions based on that theory to promote desirable change. Small changes in knowledge, attitude and efficacy may dramatically alter motivation and behavioural outcomes and the interaction of such variables can yield almost infinite potential patterns of motivation and behaviour change. Chaotic systems can be mathematically modelled but are nearly impossible to predict.

The CCC model may help to conceptualise the behaviour of some people some of the time but is insufficient by itself to explain or predict other behaviour related to addiction and transition from the addicted to the non-addicted state. Triggers for stopping the behaviour and for relapse may not conform to a set pattern and therefore to that extent the sequence of events may follow a chaotic pattern.⁹²

Undoubtedly for some people a linear change does occur and the process of change may well conform to a more logical sequence from balancing pros and cons, external pressures and decisions to change leading to setting of quit dates and progression to cessation. Any theory of behaviour change needs to incorporate a random, sudden shift of position, which may defy logic, planning, and any prediction model. A 'mixture model' of both chaotic and linear progression may be one that helps us best understand change.²²⁴

However, because chaotic systems can be mathematically modeled but can rarely be predicted the theory has very limited use in designing interventions. In many ways it becomes the equivalent of the 'error' of other models and therefore the ultimate in 'get-out' clauses.

4.5.4 Smoking cessation and the catastrophe, chaos and complexity theory (CCC)

In relation to smoking cessation, several studies have highlighted the fact that a large proportion of smokers and ex-smokers report that their quit attempts are not planned but are apparently 'spur of the moment'.^{189;214} Larabie found that 37% of current smokers reported that their most recent quit attempt had been unplanned and amongst ex-smokers 51% had not planned their successful quit attempt.²¹⁴ These results were supported by further studies by West et al.¹⁸⁹ CCC had not been used to devise their study but the authors proposed that the results were in accord with it.

Potentially these observations have profound implications for clinical work and smoking cessation research. First there is there is a fundamental instability in

the position of smokers, which may be exploited to greater benefit, and secondly quitting opportunities or 'moments' can appear at any time. It is not known what key interaction of environment or people can tip the balance for a person to quit smoking. Therefore, it makes sense to encourage smokers to both recognise and act upon 'abrupt' opportunities. Moreover, it is possible that policy makers and health professionals can extrapolate these results. Under this theory the idea that strategies should be targeted to those who are 'ready for change' would be false for at least a third of smokers. Surprisingly, smokers who make unplanned quit attempts are more likely to persist with abstinence.

These findings encourage strategies that are not 'stage related', but take the opportunities when they arise. West proposes that beliefs, past experiences, and the current situation create varying levels of 'motivational tension,' in the presence of which even quite small 'triggers' can lead to a renunciation of smoking. However if they lead to a 'plan' for later action, this may indicate a lower level of commitment to change. This concept has been incorporated in a general theory of motivation and its application to addictive behaviours.¹⁸⁹ Furthermore these authors propose that public health campaigns should focus on the '3 Ts': creating motivational tension, triggering action in smokers who are on the 'cusp' of a change, and immediate availability of treatment.

In conclusion the CCC model is a helpful conceptual tool to help explain events but because it relates to the uncertain and unpredictable, it would be inherently illogical to attempt to use it prospectively to predict the success of any particular intervention.

4.6 Generic problems with theories

*Theories in 'soft' areas of psychology lack the cumulative character of scientific knowledge. They tend neither to be refuted nor corroborated, but instead merely fade away as people lose interest*¹⁶⁴

There are plenty of problems within individual theories, making comparisons of one theory with another and with making generalisations about their utility. The problems with theories that I have encountered will be discussed under the headings of:

- Assumptions
- Study design
- Instruments – including construct and content validity
- Transferability – external validity

4.6.1 Assumptions

The social cognition and cognition theories assume that behaviour is largely under voluntary control (TPB and HBM) with predictable, planned, rational behaviour. These assumptions tend to ignore risky, involuntary or personality driven responses. Furthermore some major changes in behaviour may not only be unplanned but may be induced by apparently brief or small triggers. Addictive responses are also hard to factor-in to theories where benefits and gain are dependent on individual experience, perceptions or susceptibility rather than generalizable attitudes, experience or expectations.

4.6.2 Study design

The ideal method of testing a health behaviour theory is using experimental research design. However many studies rely on other methods including retrospective, questionnaire and cross-sectional design.

Retrospective studies rely heavily on self-reported memory of past health beliefs and behaviour which may not be reliable. Cross-sectional studies assess behaviour and belief simultaneously, cannot determine causation but only associations. Therefore experimental designs are preferable to determine the relationships between variables.

Probably the commonest method of testing theories is to use questions, which are assumed to test a certain area of cognition. Some researchers believe that the process of asking the questions may actually alter behaviour.¹⁶⁵ Application of a questionnaire may create rather than measure cognitions. Experimental research design is required to address some of these difficulties. Many of these issues would be resolved by more extensive use of prospective observational research.

Measurement and recording of behaviour or intention present some interesting challenges. There are fundamental problems with dividing the continuum of behaviour for measurement purposes. It is difficult to measure the spectrum of human behaviour or attributes with Likert scales or other intervals. Constructs or variables are often measured indirectly by questions, which may or may not give consistent or reliable results. Many 'operational definitions' of psychological

terms have been made, but they are often not generalizable. Thus there are difficulties in interpreting, understanding and comparing research and results.

4.6.3 Instruments –construct and content validity

To be useful in comparing research results and in the real world the theoretical constructs (abstract concepts of active components) held within any theory of behaviour change must not only be sufficiently defined for comparison with other studies and other theories but also be testable in a consistent manner both within the study in question and then applicable to other contexts and studies. The techniques used for testing or measuring (e.g. questionnaire) need to establish that they are consistently testing (operationalising) the constructs that are thought to be active (internal validity) and that these can be reproduced in other studies and thence to real life situations (external validity). It is not sufficient for a theory or construct to simply look reasonable (face validity), it has to have internal and external validity to be transferable to other situations in the real world of clinical understanding and intervention.

Construct validity refers to the extent to which attempts at measurement of a theoretical construct or variable (operationalisation) are 'demonstrated to be consistent with the theoretical constructs on which those operationalisations were based'.²³² An alternative definition is that 'construct validity shows how well a test links up with a set of theoretical assumptions about an abstract construct'.²³³ Construct validity is a prerequisite to external validity but the two are not the same. Furthermore some authors regard construct validity as a necessary umbrella under which other types of validity sit.

External validity involves generalizing from one study to other contexts (people, places or times), whereas construct validity involves generalizing from a programme or measures to the concept of the same programme or measures.

For example HBM proposes that perceived susceptibility and severity are two of the active theoretical construct variables. Good construct validity exists when a measure (instrument) can reliably demonstrate a good correlation between the variables that are (theoretically) active and the construct (theoretically) being measured. Some studies show fairly good correlation¹⁶² and some show only weak correlation.¹⁹⁰

Many construct measurements in behaviour theories include questionnaires with questions aimed at determining if an attribute is active. There is a need for tests of consistency and reliability to establish construct validity. There is also a great need for consistent terminology. Part of the test of consistency and thereby construct validity is the ability to translate terminology from theory to practice and between theories.

The nomological network was originally Cronbach and Meehl's view of construct validity. That is, 'in order to provide evidence that your measure has construct validity Meehl argued that you had to develop a nomological network for your measure. This network would include the theoretical framework for what you are trying to measure, an empirical framework for how you are going to measure it, and specification of the linkages among and between these two frameworks'.²³²

Content validity also sits under the umbrella of construct validity and 'seeks to establish that the item or questions are a well balanced sample of the content domain to be measured'.²³³ This is more than face validity which is fairly low in the hierarchy of features determining how well an instrument measures what it is intending to measure.

Furthermore, the lack of consistent classification or naming of different behaviours or responses presents major obstacles. For example the construct 'confidence' in trans-theoretical model is similar to Bandura's 'self-efficacy'.¹⁶¹ Some constructs are not sufficiently specific to allow them to be tested.¹⁶⁵ If a concept has not been defined in one theory then any attempt at establishing construct validity, content validity or external validity will be flawed. Therefore the instruments of measurement cannot be applied to more than one theory and any legitimate application to other studies is impossible.

The inevitable conclusion is that constructs need clear transparent definitions. How the instruments have been operationalised need to be clear and available to other researchers. In other words what questions (or other measuring tools) are used and what construct they are supposed to be testing needs to be clearly explained.

4.6.4 Transferability

External validity is essential for results to be applicable to other contexts and populations. However there may be other features which conspire against the results or theory being applied outside a particular study.

There are methodological controversies regarding the difference between idiographic and nomothetic methods in psychology. In the study of psychology, idiographic describes the study of the individual, who is seen as an entity, with properties setting him apart from other individuals. Nomothetic refers to the study of a cohort of individuals. Here the subject is seen as representing a group or population and their personality traits and behaviours. In a nutshell research in creating generalisable theories is attempting to predict the behaviour of individuals.

A number of other features may reduce the possible success of predicting behaviour. Personality differences may be due to unknown and unknowable critical events in an individual's development which cannot be predicted or generalised. Moreover the change in personality due to neurosis or depression may change the cognitive-affective voluntary actions of individuals. Differences may not be predictable or logical but simply due to luck or 'random walk' with no rational explanation.¹⁶⁴

If each and every variable is small and independent the problem of creating a useful theory would not be so problematic. However when there are large numbers of variables, which interact with each other, and contribute to idiographic development with unpredictable directions of action, the task of developing a good theory is very difficult.

There may be considerable overlap of cultural factors with other influences. Cultural differences may be mediated through related but changeable factors

(like diet, poverty, and exposure). Cultural differences may also influence mental health, reactions to situations and environment, and a person's worldview, which in turn may alter behaviour. Researchers therefore have to be careful not to make inappropriate generalisations from one context or culture to another.

Cognition theories make a link between the intention to act and the behaviour under study. Follow up studies have demonstrated that there is a large gap between intention to behave or change and actual behaviour. These gaps are often referred to as variance, which rarely lead to rejection of a theory and create difficulties with generalising the results.

Many 'operational definitions' of psychological terms have been made, but they are often not generalizable. This is particularly difficult for non-psychologists. Thus there are difficulties in interpreting and understanding research and results.

In conclusion there are many areas in which these models cause theoretical and operational difficulties. Despite these differences and difficulties there is potential to build on the work that has already been done and use any insights and ideas to better understand the rich diversity of human cognition and behaviour and use this information for improving health outcomes.

4.7 Tailored design

Although tailored design is not classified as a behaviour theory in its own right it does draw on ideas from different theories and I have chosen to include a brief description in this section. Tailoring interventions has great intuitive appeal and since the advent of computer technology has the potential to be a practical option for some interventions.

Strecher et al used a randomised study to investigate the effect of computer tailored letters to aid smoking cessation.²³⁴ Individualised letters were sent to people in the intervention group according to data collected at recruitment using the constructs of HBM as the theoretical framework. The control group were sent standard information about smoking cessation. Rates of cessation in moderate to light smokers in the intervention was improved (19% v 7%) significantly compared to the control group ($P < 0.5$)ⁱ.

Taking advantage of modern computer technology the Etter et al tested the hypothesis that computer-tailored counselling letters (and stage matched booklets) would promote better intentions to quit than non-tailored advice.¹⁵⁶ They based their intervention on a combination of behaviour models including the stage of change (TTM), and the theory of planned behaviour (TPB). Nearly 3000 smokers were recruited by random mailing of 20 000 addresses from a population register. Their power calculation required 2000 participants and they

ⁱ No confidence intervals were given in the paper.

estimated a 10% response rate to the mailing. Respondents were randomised into intervention and control groups. Personalised letters sent to the intervention group used the level of tobacco dependence, attitudes toward smoking, self-efficacy, and intention to use nicotine replacement therapy.¹⁵⁶ They demonstrated a significantly improved rate of cessation in the intervention group in all categories (5.8% v 2.2%) ($P= 0.001$). Abstinence was not verified by biochemical testing. A large proportion of the participants were not considering quitting at the start of the study and even those in the group of pre-contemplators fared better with tailored information (3.8% v 0.8%).

Computer technology allows for personalised relevant targeted information to be given to an individual smoker. There is some evidence that this targeted approach is a useful method to help some smokers to quit.

5 Methodology

In order to evaluate the impact of lung age information we need to measure key variables. In this section I outline some of the common instruments in use and discuss their advantages and disadvantages. I also seek to explain the reasons for choosing the instruments used in this study.

5.1 How to measure smoking status

It is important to decide on the meaning of smoking cessation and what constitutes a successful intervention. The Cochrane study of NRT interventions aimed to select studies which had biochemical validation of smoking cessation.¹⁴¹ Within their review the definitions of abstinence varied. They found that the meaning of sustained abstinence included at least three different interpretations: continuous abstinence without one cigarette since the quit day, repeated point prevalence at repeated telephone or direct contact, and self reported abstinence for a prolonged period. A quarter of the studies in that review only reported the point prevalence at the longest period of follow up. The reviewers stated that they preferred the use of sustained cessation rather than point prevalence and excluded studies, which did not have cessation data for at least six months following the intervention.

There are problems whichever measure of cessation is used. Point prevalence is not necessarily stable; continuous abstinence inevitably reduces with time

and measuring prolonged abstinence requires lengthy follow up. The desirability of using point prevalence is that it is the only state that can be confirmed biochemically. Repeated point prevalence with confirmation of cessation by biochemical monitoring helps to determine if abstinence is consistent but requires more frequent contact and the smoker-clinician contact may be seen as part of the intervention. There is currently no technique available for continuous monitoring of smoking status to give a reliable measure of sustained abstinence.

The need for biochemical validation is controversial but often necessary to convince peers and journal editors as well as funding institutions. Indeed satisfying the funding agency that smoking cessation rates would be biochemically confirmed was essential to the success of the application for a research grant. The accuracy of self-reported abstinence is controversial. Some studies claim that self-reporting is reliable and accurate in most cases and some insist that biochemical validation is unnecessary.²³⁵ Several other studies have demonstrated that deception rates may be as high as one in four. In other words 25% of people who claimed to have stopped smoking will have significant levels of cotinine indicating continued cigarette consumption.¹⁵³ The large discrepancy between self-reporting and biochemical verification in the lung age study results supports the notion that validation is necessary in research and is discussed elsewhere in this thesis (see Table 6, p. 196)

Smoking or smoking cessation can be measured by two main biochemical

methods; measurement of breakdown products of nicotine (in blood, urine or saliva) and measurement of carbon monoxide (in blood or exhaled air).

5.1.1 Biochemical measures of smoking

Among biochemical methods of confirmation the use of a biochemical breakdown product of nicotine called cotinine is the most accurate but the tests are intrusive and expensive. Cotinine is a metabolite of nicotine, and it or the hydroxyl metabolites of cotinine can be measured in urine, blood or saliva. Although cotinine levels can be an accurate way of determining smoking status the use of nicotine gum or patches will also result in raised levels of cotinine and therefore smoking status can only be verified through simultaneous use of a carbon monoxide monitor. Although some vegetables and other foods contain nicotine, huge amounts would need to be eaten to invalidate a cotinine test for active smoking, but passive smoking may create false positives.²³⁶⁻²³⁸

The optimum cotinine cut off point to distinguish smokers from non-smokers is 14.2 ng/ml, which correctly classifies 99% percent of non-smokers and 96% percent of smokers. The half-life of cotinine is approximately 20 hours and therefore the test can detect most people who have smoked a cigarette within the past 48 hours. As already mentioned the disadvantage of this technique is that continued use of nicotine replacement therapy also gives positive results. Therefore simultaneous recording of current use of NRT is vital for interpretation of the results (combined with concurrent measurement of exhaled carbon monoxide).

Among the tests for cotinine, I chose to use the saliva cotinine test for my study.

This seemed to have a number of clear advantages. Collecting saliva for analysis is the least invasive and intrusive, easy to collect and despatch, and the instructions for use were easy to learn and teach. An independent nurse was instructed to personally witness the collection process without having the need for special skills (of phlebotomy).

To ensure the independence, validity and reliability of the results I chose a registered independent laboratory offering a postal service. Specimens were processed by ABS Laboratories, Medical Toxicology Unit, Wardalls Grove, Avonley Rd, London, SE14 5ER. I also chose two independent highly qualified registered nurses to collect the samples by home visits where feasible to maximise the collection of samples. One was a district nurse and the other had trained as a respiratory nurse whose job involved home assessment of patients with chronic respiratory disease. Neither of the nurses was involved with other aspects of the research project.

Although nicotine breakdown products are not routinely measured in clinical practice (as the tests are expensive and results are not rapidly available) carbon monoxide measurements are quick and easy to make after a modest capital outlay for the measuring device and are routinely used in smoking cessation clinics to confirm smoking status. Carbon monoxide can also be measured in blood but this is more invasive, needs phlebotomy skills and does not give immediate results. The potential advantages of blood measurement would be that it does not rely on the breathing and coordination skills of the participant and that results are independently confirmed. The advantage of measuring

carbon monoxide in exhaled air is that it can give immediate information to the examiner and feedback to the patient.

Controversially the NHS continues to use the four week smoking cessation figures as the measure of success with point prevalence either self reported or validated with the measurement of exhaled carbon monoxide.²³⁹ There have been calls for clearer standards for reporting within the health service which would enable meaningful comparisons between services and research.²⁴⁰

There are two main reasons to measure smoking levels in this study. The first is to confirm the veracity of the claim of a participant that they have stopped smoking so that independent confirmation of systemic nicotine can be tested and secondly to use both the levels of carbon monoxide in exhaled air and systemic nicotine in combination to determine if raised cotinine levels are due to nicotine replacement therapy. I used carbon monoxide monitors for initial confirmation of cessation at the twelve-month examination and during saliva collection for cotinine testing. I chose the model in current use by our local primary care trust and issued for regular use in the smoking cessation clinics. The device is simple to use and made by a reputable medical instrument company (Smoke Check – catalogue number SC01).²⁴¹ This model has a carbon monoxide range of zero to 500 per million and a sensitivity of one part per million.

Using this model had three clear advantages. Firstly, they were already

accepted within the NHS as complying with quality standards. Second, it made economic sense to use equipment already available and in use. Finally, the clinical staff were already familiar with the equipment and did not require additional training.

5.2 *Should nicotine dependence be measured and if so how?*

Quitting success has been associated with levels of nicotine dependence. Therefore it can be a useful measure when comparing groups within a study and when comparing studies with one another. Furthermore, it is assumed that identifying those with greater nicotine dependence will help target treatment.

The Fagerström tolerance questionnaire (FTQ), Fagerström Test for Nicotine Dependence (FTND), and the Heaviness of Smoking Index (HSI) are all widely used measures of nicotine dependence in smoking research. FTND and HSI are similar in their ability to predict smoking cessation to a small degree. Both the FTQ and FTND have strong internal validity (high retest reliability).

The three measures of nicotine dependence are inter-related. The FTQ was the first of the three and is longer than the other two. Many reviewers believe that it should make way to the others.²⁴²

5.2.1 FTQ (Fagerström tolerance questionnaire)

The development of the tolerance questionnaire relied on the correlation between physiological measures of tolerance of nicotine with physical dependence during smoking and withdrawal. The aim was to develop a way of measuring physical dependence.

Box 4 Fagerström tolerance questionnaire (FTQ)

1. How many cigarettes per day (cpd) do you smoke?

Score: 0= 15 or less cpd, 1= 16 to 25 cpd, 2= 26+ cpd

2. What brand do you smoke?

Nicotine yield – score: 0= 0.9mg or less, 1= 1-1.2 mg, 2=1.3mg +

3. Do you inhale?

Score: 0=Never, 1= Sometimes, 2= Always

4. Do you smoke more during the morning than during the rest of the day?

Score: 0=No, 1=Yes

5. How soon after you wake up do you smoke your first cigarette?

Score: 1= within 30 minutes.

6. Which cigarette would you hate to give up‘?

Score: 1 for ‘First one in the morning’.

7. Do you find it difficult to refrain from smoking in places where it is forbidden, e.g. in church, at the library, cinema?

Score: 1 for yes

8. Do you smoke if you are so ill that you are in bed most of the day?

Score: 1 for yes

Scoring range is 0-11 points. Eleven indicates maximum dependence

With regard to the FTQ presented above there are a number of key points as follows:²⁴³

- They assumed that brands which contain a higher dose of nicotine are consumed by those smokers with more physical dependence.

- Inhalation makes absorption of nicotine more efficient and is likely to be linked with dependence (Question 3).
- Dependent individuals will need nicotine when levels are low in the morning (Question 4, 5 and 6).
- The urge to smoke in inappropriate places or when ill points to less control and more dependence (Questions 7 and 8).

The questions have face validity on the basis of widespread views, assumptions and observations about smoking and dependence at that time.

Separating the physiological and psychological dependence using heart rate and body temperature requires good quality scientific methods such as using randomised double blind, placebo controlled trials. Most of the trials have been small scale non-blinded and not randomised.²⁴⁴ Although the tolerance scale had initial face validity the correlation between scores and withdrawal symptoms is weak.²⁴⁴ However the FTQ does correlate well with other biomarkers of the effects of smoking such as cotinine levels and carbon monoxide levels.²⁴⁴ Other investigators have found no correlation between the FTQ and most of the withdrawal symptoms including irritability, anxiety, restlessness, hunger, impatience, somatic complaints and insomnia. There was significant correlation between FTQ score and craving and also with difficulty concentrating.²⁴⁵

A stated function of the FTQ is to predict the success of smoking cessation and to target those smokers with high dependency who cannot quit without help. There is some evidence to support the idea that high nicotine dependent smokers do better with nicotine replacement than less dependent people.²⁴⁶

However low dependent people will do better with nicotine replacement than with placebo alone.

5.2.2 Fagerström Test for Nicotine Dependence (FTND)

The Fagerström Test for Nicotine Dependence (FTND) (see Box 5, p. 153) was developed from and has largely replaced the FTQ. Because most smokers inhale and nicotine rating of cigarettes is an unreliable indicator of absorption of nicotine (as measured by biochemical means), several questions contained in the FTQ were discarded. Revision of the scoring system for time to first cigarette and the number smoked per day improved the scale.²⁴⁷ The FTND is shorter than the FTQ (six versus eight questions) and simpler in application and has a growing body of literature to support its use although the association between nicotine dependence level measured by the scale and withdrawal symptoms remains small.²⁴⁸

Higher test scores are associated with carbon monoxide levels but high-scoring smokers are not clearly differentiated from those with mid-range scores.²⁴²

Box 5 Fagerström Test for Nicotine Dependence

FTND

The score for each answer is in brackets (). Add up the scores and compare with the limits at the foot of the page.

1. How soon after you wake up do you have your first cigarette?

- A. Within 5 minutes (3)
- B. 6-30 minutes (2)
- C. 31-60 minutes (1)
- D. After 60 minutes (0)

2. Do you find it difficult to refrain from smoking in places where it is forbidden, e.g., in church, the library, the cinema, etc?

- A. Yes (1)
- B. No (0)

3. Which cigarette would you hate most to give up?

- A. The first one in the morning (1)
- B. All others (0)

4. How many cigarettes do you smoke per day?

- A. 10 or fewer (0)
- B. 11-20 (1)
- C. 21-30 (2)
- D. 31 or more (3)

5. Do you smoke more often during the first hours after waking than during the rest of the day?

- A. Yes (1)
- B. No (0)

6. Do you smoke even if you are so ill that you are in bed most of the day?

- A. Yes (1)
- B. No (0)

Score: 7 to 10 points = highly dependent on nicotine

4 to 6 points = moderately dependent on nicotine

less than 4 points = less dependent.

Unfortunately there is poor correlation between this and other widely used measures of dependence on nicotine such as the Diagnostic and Statistical Manual of Mental Disorders (DSM III-R criteria). The implications of these differences will be discussed in the next section.²⁴⁹

5.2.3 Heaviness of Smoking Index (HSI)

The HIS was developed from the FTQ and the FTND in 1989 after the realisation that most of the difference in scores comes from just two of the items in the other two scoring systems.²⁴⁷

The heaviness of smoking index is created as the sum of the number of cigarettes smoker per day and the time to first cigarette. HSI scores range from 0–6 and are calculated by summing the points for time to first cigarette (TTF) after waking and number of cigarettes smoked per day (CPD).

Box 6 Heaviness of Smoking Index (HSI)

1. Time to first cigarette (tff)

Score: < 5 mins tff = 3 points

6–30 mins tff = 2 points

31–60 mins tff = 1 point

> 60 mins tff = 0 points

2. Cigarettes smoked per day (cpd)

On average, how many cigarettes do you smoke each day, including both factory-made and roll-your own cigarettes?

Score: 1–10 cpd = 0 points

11–20 cpd = 1 point

21–30 cpd = 2 points

> 31 cpd = 3 points

Higher HSI scores indicate more dependence on nicotine.

The HSI is positively associated with nicotine dependence, and functions as well as the FTND. A prospective study, of nearly 1000 smokers, of the ability of the three self-reported tests (FTQ, FTND and the HSI) to predict levels of carbon monoxide at the outset and the likelihood of cessation after group cessation were statistically significant.²⁴² The HSI has gained in popularity and use in the field of smoking research and has been applied in some recent large multi-centre cross-sectional studies.²⁰ However, in the mid range of HSI values in a large survey conducted in the U.K. (2006), there was a lack of an association between nicotine dependence and quit success compared to that found in other countries (e.g. Canada).⁸⁰

5.2.4 Craving

The subject of cravings is controversial as it refers to subjective feelings, which are thought to relate to nicotine dependency. There is little consensus about how best to define or assess cravings.²⁵⁰

Box 7 Questionnaire of smoking urges

Smoking urges²⁵⁰

1. I have a strong desire for a cigarette right now.
2. If it were possible I would smoke right now.
3. All I want now is a cigarette.
4. I have an urge for a cigarette.
5. I crave a cigarette right now.

Despite the controversies, withdrawal and craving may be important as potential

predictors of relapse, or as markers of treatment effects. Multiple questions with scales for different responses have been developed. Non of them have come into regular and widely accepted use.^{216;250} However, the assessment of *cravings* may be operationalized by using a single question :

On a scale of 1- 10 how strong are your cravings right now?

Some investigators have found this form as reliable and valid as longer scales and demonstrate ability to predict smoking behaviour.²⁵⁰ There are several longer scales used in research including the questionnaire of smoking urges, which asks respondents to indicate on a line (from strongly agree to strongly disagree) the strength of their agreement with the five items in Box 7 (p. 155) and a ten point craving scale which is similar.²¹⁶

5.2.5 Critique of measures of nicotine dependence

The development of reliable scales of nicotine dependence has potential use in both clinical practice and research. In theory the measurement of dependence should help practitioners to target those who need pharmacological help and then to titrate doses of nicotine replacement in accordance with degree of addiction. Nicotine replacement treatment (NRT) manufacturers only recommend the use of their products when smokers are consuming ten cigarettes or more per day. However the evidence for use of titration is not strong and NICE technical guidelines on NRT do not set any lower limits of smoking dependence for the use of NRT.¹³⁷ Therefore currently, the main use of measures of dependence is in research.

The implications of the differences in scoring with different systems are that estimates of the prevalence of nicotine dependence in the general population

may vary depending on which instrument is used.²⁴⁹ Some research studies enroll participants using criteria based on the level of dependence. If different studies use different tools the results cannot be directly compared or used in meta-analysis. Any of the individual tools for measurement can be and are commonly used for interventional studies to confirm similarity of control and intervention groups and successful randomisation.

5.2.6 Rationale for non-inclusion

A measure of nicotine dependence was not included in the lung age RCT. Even though measures of nicotine dependence are much more strongly associated with cessation than measures of motivation, and nicotine dependence is regarded as a major factor predicting long-term cessation in smokers,²⁷ this study was not designed to identify associations between cessation and dependence.

Furthermore, due to the poor correlation of nicotine dependence scores with each other or with particular interventions it is not yet reasonable to target particular groups of smokers or individuals. Our research study was not designed to target a particular group, indeed the recruitment process was deliberately aimed at being as inclusive of all smokers over the age of 35 and reducing the exclusions to a minimum to reflect the primary care population of smokers and not simply those seeking help with cessation.

In hindsight there would have been some merit in collecting data on nicotine and this will also be discussed in Section 8.2 (see p. 207). The extra data would have added another tier of information to strengthen the evidence that

randomisation had been successful. It may also have given some data about any correlation of lung damage and nicotine dependence but this was not the aim of the study, which was neither powered nor designed to test that theory. Also it would have been a distraction from the main purpose and taken extra time during recruitment and collection of the important core data.

5.3 Should psychological variables be measured and if so how?

Psychological factors influencing behaviour change have been extensively discussed in the chapter on behaviour change theories (see Section 4, p. 89). In order to avoid repetition, here I will discuss three important areas of psychology that I believe are most relevant to this study and refer back to previous sections where necessary. The three factors I will discuss in this section on measuring psychological factors are:

- Motivation related to intention
- Self-efficacy
- Perceptions of risk to health

Psychological factors are thought to have an important influence at every stage of smoking behaviour from initiation through to decisions to quit and factors in relapse. Therefore, investigators should consider their impact at every stage of the research process from designing interventions through to interpretation of results.

The detail of the development, testing and validation of psychological tools is beyond the scope of this thesis. However it is of interest to outline some of the psychological factors that are most likely to be active and influential in smoking behaviour change in the context of this study of lung age.

5.3.1 Measuring motivation related to intention to quit.

In national surveys the majority (~70%) of smokers say they want to quit,⁷⁹ but there may be barriers from turning this intention or motivation into reality. One simple schema to assess this in a qualitative way is the grid below indicating the relationship between motivation and dependence.

Table 4 Motivation, dependence and intervention goals

Motivation	Dependence	Characterised by	Primary intervention goal
Low	Low	Unlikely to stop but could do without help	Increase motivation
Low	High	Unlikely to stop	Initially to increase motivation to make receptive to treatment for dependence
High	Low	Likely to stop with minimal help	To trigger a quit attempt
High	High	Unlikely to stop without help but would benefit from treatment	Engage smoker in treatment

(Table adapted from West⁷⁹)

When considering measurement of motivation about smoking and cessation the

different meanings of the term need to be considered. In terms of the study of motivation it may be regarded as the study of movement or action.²⁵¹ This may be passive in the sense of being pulled along by a desire or actively being pushed along by a drive e.g. the desire for better health. Different models of motivation by necessity are assessed and measured in different ways

5.3.2 Maslow's hierarchy

According to Maslow's hierarchy of needs, people have a desire or are motivated to move from one level in the hierarchy to another.²⁵² This starts with the fulfilment of basic needs of life including warmth, food, clothing and shelter. Once each level is attained and satisfied then an innate need to reach the next level becomes the motivator.

In the words of Maslow: 'Man is a perpetually wanting animal. Also, no need or drive can be treated as if it were isolated or discrete; every drive is related to the state of satisfaction or dissatisfaction of other drives'.²⁵³ Therefore once a lower level is attained then theory states that there is a motivation to move to higher levels through those of safety and love and esteem to self-actualisation. Paradoxically, despite self-actualisation being the pinnacle of the hierarchy, apparently this does not bring about satisfaction or eliminate the drive for change.

Motivation theory is not the same as behaviour theory. The motivations that Maslow describes are only one class of determinant of behaviour. Although behaviour is motivated it is also determined by other forces of biology, culture and circumstance.²⁵³ Although the hierarchy of needs has been widely adopted

in the processes of change in books and popular culture, and also in the realms of personal and professional development, it has not found a strong niche in research into smoking. The framework may fit into the field of smoking and cessation in a number of different ways. First, a person who seeks satiation of hunger may really be seeking more for comfort, or dependence. In contrast, a person may satisfy hunger or believe they are reducing stress by smoking a cigarette. Secondly, when a person is seeking to quit smoking they may be motivated by a desire to reduce threat to health thereby improving *security* in the Maslow hierarchy. Discovering how people view their smoking and their need to continue may give insights into how they can approach quitting and what factors may lead to relapse.

A problem arises when conflicting cognitions come into play. A message that 'smoking is bad for you long term' may be come up against the desire for immediate short term pleasure from a cigarette. The conflict or dissonance between the two cognitions leads to stress and anxiety which can be expelled by rejecting one of the messages or changing behaviour.^{251;254}

Although the theoretical framework is of use in qualitative understanding of some behaviour and may be used as part of the underpinning rationale for some forms of therapy such as motivational interviewing (see p. 70), I have been unable to find any smoking cessation research studies which have used scales of motivation based on Maslow's hierarchy and cannot conceive of a way that this could be adapted for use in this purpose.

5.3.3 Readiness and stage

Changes in motivation may be conceptualized as a readiness to change as described in motivational interviewing.^{142;255} Proponents of motivational interviewing describe motivation as simply the probability that a person will enter into and adhere to a specific change strategy. The role of the therapist is to facilitate and support. This approach assumes a phenomenological, humanistic approach of self-actualisation.²⁵⁶ This is in contrast to traditional cognitive theories of motivation and any confrontational approach by the interviewer.²⁵⁶ The phenomenological approach to change is based on a view that man has choice and that change is intentional. Accordingly there is potential for growth and change as a process of self-actualisation.

The readiness to change ladder, has face validity and consists of eleven response options on a continuum ranging from 'not at all considering quitting smoking in the near future' to 'already have quit smoking'.²⁵⁷ Ladder scores have been found to predict subsequent participation in programs designed to educate smokers about their smoking habit and its risks. However, the ladder scores do not predict abstinence confirmed by biochemical testing.

The ladder has been associated with intention to quit, nicotine dependence and numbers of previous quit attempts.²¹⁶ As discussed in the chapter on behaviour change this adds weight to the conclusion that intention to quit is a poor predictor of successful quitting.

Although the popularity and standing of the transtheoretical model and the

concept of stages is waning, many studies continue to use the idea of linking intention and motivation. For example a recent study in older smokers used the following question²⁵⁸:

‘Are you planning to quit smoking within the next month, next 6 months, sometime in the future (beyond 6 months) or not planning to quit?’

Responses were dichotomized into planning to quit versus not planning to quit, without overtly using TTM as a theoretical framework or presenting evidence that the question is a valid and reliable way of testing motivation. This seems to me a clumsy way of using what in essence is the same system.

Changes in motivation may be conceptualized as a movement through stages as described in the trans-theoretical model (TTM).²¹¹ With regard to stages of change it is believed that most people presenting for treatment are by definition in the preparation stage. However there is no consistent evidence that these stages predict movement nor are they stable.²⁵⁹ As many as 30% of smokers will alter their stated intention within a 30 day period.²⁵⁹ The trans-theoretical model and stages of change is discussed in detail in Section 4.4 (p. 116).

5.3.4 Rationale for choice of measuring tool

The decision to use the trans-theoretical stages of change in this study was based on the pragmatic need for a simple and quick, staging and readiness to change, measure. The format used by the trans-theoretical model undoubtedly fulfils these two criteria.

At the time of the start of this project the trans-theoretical model of stages was commonly used in quit research. In particular, many previous studies exploring

the use of spirometry or biomarkers to promote quitting, have used a self reported 'intention to quit' measure based on the trans-theoretical stages questions or similar brief questioning.^{36;155;217;260} Even very recent large multi-centre international studies of predictive factors of smoking have used intention to quit based on almost identical questions used in this model.²⁷ In contrast, some key studies make no mention of a motivation or intention to quit measure in their methods or results.³⁵ A minority, however, use a surrogate measure of past motivation called 'previous quit attempts', but no detail is given about the period of time this refers to, and how it is measured or validated.^{148;151;157;217}

The main criticisms of the widespread use of the trans-theoretical stages of change classification have become evident since the research protocol for this study was developed (see Section 4.4.3, p. 123).²³¹ Even new studies of the psychological mechanisms of confrontational smoking cessation interventions using spirometry are not using sophisticated or widely established measures of motivation e.g. a study by Kotz (in press) used the question 'how important do you find it to stop smoking on a scale of 0 (not important at all) through 10 (very important)?'²⁶¹

Despite the criticisms of the trans-theoretical model no clear consensus has emerged to support an alternative scoring system of motivation to quit smoking, for research or clinical practice or for targeting interventions. Even though this model is not ideal, is neither stable nor reliable method of establishing readiness to receive treatment, at least some comparisons can be made with previous research on using spirometry to promote smoking cessation. All else

being equal the use of stages of change in this trial supports the conclusion that randomisation was successful and similar rates of responses were obtained from both the control and intervention groups. It also supported the notion that the participants were not exclusively from a group of smokers on the verge of quitting. It is logical to assume that smokers who voluntarily attend smoking cessation programmes are motivated and in a state of readiness to change their behaviour. Some research programmes' recruitment methods result in a high proportion of participants who are already 'planning to quit', or even overtly exclude those who are not planning to quit¹⁵⁹ and therefore any results of successful cessation are biased by recruitment or selection method. For example, among the over 400 participants, in a randomised trial of biofeedback on lung cancer risk, only 3%^j of the participants were not considering stopping smoking within the next six months.²¹⁸

In conclusion, despite its flaws, one clear benefit of using the blunt instrument of stage of change (TTM) is that the information may help determine whether or not comparisons can be made between studies using the same measures of motivation whereas if no measure is available there will be greater doubts about usefulness of making comparisons.

^j Pre-contemplative 3%, contemplation stage 57%, preparation stage 40%²¹⁸

5.4 Other measures of cognition

The concept of self-efficacy is included in most of the cognition and social cognition models of behaviour change (see Section 4.1.3, p. 92). Self-efficacy conceptualises an individual's belief in their ability to perform a particular task or behaviour²⁶² and is probably important in determining the success of smoking cessation^{263;264} and relapse is consistently predicted by measures of self-efficacy.²⁶⁵ Those who have been smoking a long time are more likely to be less confident about stopping.²⁵⁸ Differences in self-efficacy have been implicated as part of the reason that cessation rates are lower among lower socio-economic groups.²⁰

Measurement of self-efficacy is usually through questions about confidence. There is a wide range of tools available to researchers, which attempt to measure self-efficacy. In some studies measurement is brief and operationalized with a single question e.g.

1. 'If you decided to give up smoking completely in the next 6 months, how sure are you that you would succeed?' (Options of -Not at all sure/slightly sure/ moderately sure/ very sure/ extremely sure)^{20;80;258}
2. 'How confident are you that you could quit smoking for good? (Options 0= not at all, 2= a little, 3= very much, 4= extremely)^{217;266}

The most recent large prospective cross-sectional study, in four developed countries, suggests that most of the variables tested predicted intention or attempts to change rather than to the success of attempts.⁸⁰ Self-efficacy is the

possible exception but the correlation is not strong. This is in keeping with the evidence presented under the chapter on health belief models (see Section 4.3) where there is a serious gap between intention to change and successful changes in behaviour. Measures of nicotine dependence were the only measures in that study which predicted both quit attempts and quitting, which suggests that interventions that focus on dependence are preferable to interventions that only focus on expectancy-based variables.

Alternatively more complex instruments can be used such as the confidence questionnaire. The original questionnaire is far too lengthy and even the fourteen item shortened form (form S), later introduced is too long for clinical contexts and most research studies.^{216;267} Efficacy means more than the initial confidence about quitting; it also encapsulates the confidence and ability to remain abstinent.

The relapse situation efficacy questionnaire (RSEQ) aims to measure vulnerability depending on context and situation.^{216;268} The questionnaire asks the respondent to rate their confidence at not smoking under a variety of conditions on a scale of 'not at all confident' to 'extremely confident' (1 to 4). Excerpts from the RSEQ can be seen below.

1. 'How confident are you that you can resist the temptation to smoke when you are: restless, tired, happy, irritable, sleepy, tense, hungry etc'?
- Each of these categories are answered separately.
2. 'How confident are you that you can resist the temptation to smoke

- when you are feeling very bad, bad...to very good’?
3. ‘How confident are you that you can resist the temptation to smoke when your arousal or energy level is very low/ low/ to /very high?’
 4. ‘How confident are you that you can resist the temptation to smoke when you are eating and drinking?’

A number of different scenarios are outlined concerning type and timing of consumption of food and alcohol to help the informant to understand and answer the questions.

A measure of confidence was not measured in the lung age study and the implications of that decision are discussed further in Section 5.5.1 (p. 169) and under the discussion of the weaknesses of the study (Section 8.2.2, p. 207).

5.5 *Should attitudes and beliefs about smoking be measured?*

5.5.1 What is an attitude?

In terms of measurement, the consensus amongst most researchers is that an attitude is a state of readiness and a tendency to respond in a certain way when faced with certain stimuli. Most are dormant and become apparent in speech or behaviour when the object of the attitude is perceived.²³³ Attitudes are reinforced by beliefs (the cognitive component) and may be accompanied by strong emotion, which may lead to particular intention or action. They are abstract and may not fit into any logical or recognisable pattern or mixture.²³³ Attitudes are said to be part of a mix of values, beliefs and feelings.

Attitude scaling is a common method of measurement used in surveys. They are not designed to yield insights into individual cases and according to Oppenheim they should not be used as a clinical instrument.²³³ Their main function is to place people on a continuum in relation to one another but cannot measure them in absolute terms.²³³ In terms of research they may be used to explore associations between attitudes and differences in behaviour. For example, research has found associations between concern on the part of smokers about their susceptibility to serious illness and their attempts to quit smoking and reduction of cigarettes smoked.²⁰⁸

Any behaviour may be graded on an arbitrary scale by asking a series of questions about attitude e.g. as conceptualised by TPB (see p. 98). This may

include-

- a person's evaluation of the behaviour,
- their perceived risk of diseases associated with that behaviour and
- measures of changed perceptions of risk depending on the message or intervention under investigation.

5.5.2 How can attitudes be measured?

One important example of the use of scales in exploring the attitude variables active in smokers is as follows (see Box 8, p. 170 and Section 4.2, p 98):

‘My not smoking (continuing to smoke) over the next six months would be’:		
Harmful	-3, -2, -1, 0, +1, +2, +3	Beneficial
Unpleasant	-3, -2, -1, 0, +1, +2, +3	Pleasant
Un-enjoyable	-3, -2, -1, 0, +1, +2, +3	Pleasant
Bad	-3, -2, -1, 0, +1, +2, +3	Good
Foolish	-3, -2, -1, 0, +1, +2, +3	Wise
(Adapted from Norman et al) ²⁶⁹		

Box 8 Measure of attitude towards not smoking

‘Respondents attitude towards not smoking over the next six months was measured using three semantic differential scales (i.e. bad-good, harmful-beneficial, foolish-wise) scored -3 to +3. The questionnaire also included a measure of perceived susceptibility. Respondents were presented with a list of seven smoking related health problems (e.g. lung

cancer, bronchitis) and asked to answer the question':

'How likely do you think it might be that you will develop any of the following problems in the future if you continue to smoke?' on a seven point scale, scored -3 to +3.²⁶⁹

One example of how concern about health may be operationalized is as follows in the study by Lipkus 2007⁵⁶:

Participants were asked a question with four parts.

'To what extent do you feel worried that smoking is currently '

1. harming your lungs?
2. harming heart and circulatory system (e.g. increasing blood pressure)?
3. causing breathing problems (e.g. coughing, shortness of breath, wheezing or asthma)?
4. causing more colds?

Response options ranged from 1 (not at all) to 7 (extremely).

A similar but not identical set of questions were used in a large population survey in four continents as follows.⁸⁰ They called this 'worries about health and quality of life': This variable was created based on smokers' responses to two questions at baseline:

- (1) 'How worried are you, if at all, that smoking will lower your quality of life in the future?'
- (2) 'How worried are you, if at all, that smoking will damage your health in the future?'

Categories of the answers include:

not at all worried, a little worried, moderately worried, very worried.

Each variable was coded as a continuous measure from 1 (weak) to 4 (strong) and the average of the two measures was used in analyses.

The same study also tested the outcome expectancy of quitting:

At baseline survey, smokers were asked,

“How much do you think you would benefit from health and other gains if you were to quit smoking permanently in the next 6 months?” Categories of the answer include:

not at all, slightly, moderately, very much, and extremely.

The variable was coded as a continuous measure from 1 (weak) to 5 (strong).⁸⁰

There is some recent support for the notion that confronting smokers with previously undiagnosed airways obstruction with the results of spirometry increase risk perceptions and self-efficacy, and decreases risk denial (self-exempting beliefs) in already motivated participants.²⁶¹ These changes in mediators are associated with a higher likelihood of smoking cessation. Risk perception was measured using two simple questions:

1. ‘How high do you estimate your risk of getting a serious disease at a later age when you do not stop smoking?’ (1=very low to 5 very high)
2. ‘How high do you estimate your risk of getting a serious disease within the next ten years when you do not stop smoking?’ (1=very low to 5 very high)

Self-exempting beliefs are commonly found among smokers.^{270;271} According to this idea if a smoker believes he is susceptible to the ill effects of smoking but continues to smoke this creates cognitive dissonance which creates anxiety. The concept of self-exemption implies that the individual reduces the anxiety by believing something else to create risk denial e.g. that smoking is not really as harmful as reported. Therefore, self-exempting beliefs are thought to have the potential to interfere with smoking cessation. Researchers have tried to measure these self-exempting beliefs by using questions in a number of ways. There is some overlap in the selection of questions used in different studies to test exempting beliefs as follows:

Respondents were asked to rate the following statements-

Study 1. 'Smoking is no more dangerous than breathing polluted air in urban areas',

Study 2. 'Some people can smoke their whole life and never get sick,'

Both used a four point Likert scale (strongly agree, agree, disagree, strongly disagree).

When quoted in the literature these tools are often adapted from use in other contexts (e.g. attitudes to increased exercise, health screening, or eating more fruit). The examples given above are important in that they highlight two problems with testing attitude in smoking research. First, among the large numbers of studies of attitudes, there is a lack of standardisation of questions and secondly there is a lack of evidence of validation available in the literature.

5.5.3 Rationale for not using attitude or cognitive factors

A pragmatic decision was made not to include a measure of psychological factors (including self-efficacy or perceived risk or perceived benefit from quitting) in this research project. In the context of primary care research the data collection tools have to be valid and concise to be acceptable to both the participating practices and individuals. Evidence available at the time of the development of the study did not suggest that the concept of self-efficacy was implicated in determining smoking behaviour in the context of knowledge of spirometry results or lung age. The main aim of initial data collection was confirmation that the two randomised groups were similar at baseline. Extending the baseline tests on the basis of an unfounded speculation that self-efficacy would be a major factor influencing the reaction to lung age was not part of the theory. Furthermore, during the initial literature review (even prior to the Cochrane review of biomarkers and smoking cessation)¹¹⁵ I did not identify any randomised or cross-sectional studies which had used psychological measures of self-efficacy as part of the baseline data collection and therefore no precedent had been set and no comparisons with previous studies could be made even if such a measure had been included.

However, in the light of the results it would have been interesting to have recorded some measures of risk perception both before spirometry and after explanation of the results. Reactions to the information given and how this had change perceptions and intentions would have been interesting. There is only one published research paper on the effects of lung age on perceived health risk and desire to quit. This was a small pilot project among American college

students.⁵⁶ They used a number of tools to measure health behaviour risk and emotional reaction to lung age information based on the health belief model. There are too many problems with the Lipkus et al. (2007)⁵⁶ study to make any meaningful comments about the 'results' or make generalisations. For example, based on population surveys, it is highly improbable that students of this age would show any abnormality on spirometry after an average of only four pack-years of smoking but remarkably 75% of those tested had abnormal results (average lung age 35 versus average age 20), numbers were too small and no power calculation was done prior to recruitment to come to any meaningful conclusions. However they have made inroads into how to test a hypothesis about the mechanism of action of giving lung age as the intervention.

I had some expectation that poorer lung age would lead to bigger likelihood of changed behaviour and some fears that good results might lead to indifference to change. However, to have immediately measured the psychological effects of the information may itself have been regarded as part of the intervention and influenced perception and intentions (see Section 4.6, p. 134).¹⁶⁵ Moreover, this would have been rather impractical as the intervention included written postal individual feedback and therefore any questionnaire about changes in self-exempting belief would have to be administered after the participant had read the letter. Any extra contact for data collection would have added complexity to the study and was therefore not considered.

In summary, although psychological data would have been interesting, the randomised controlled trial was not designed with a behavioural theory in mind

and was not intended to analyse the intricacies of psychological forces related to lung age. Further research is required to explore the mechanism of action of the success of lung age as a smoking cessation tool. Further discussion about psychological measures will be included in the section on the weaknesses of the study (p. 205) and ideas for further research (see Section 8.3, p. 210).

5.6 How to measure impact of smoking

For the purposes of this study I was primarily interested in selecting a widely used, validated questionnaire that could measure the impact of smoking on the respiratory health of participants so that the control and intervention groups could be compared at baseline. I anticipated that this would have two important functions necessary for this study. First it would be supportive evidence of successful randomisation. Secondly, it would be another measure of the similarity of the two randomised groups, in addition to the objective measures of lung function and demographic data collected.

Objective measures of lung function (e.g.FEV1) do not necessarily correlate with the impact of lung function on daily life, as patients tend to adapt gradually to their limitations. Significant airways obstruction may be present before the individual is aware of any problem.³² Treatment of airways disease is directed towards improving patients' health and wellbeing. Measurements of airways function do not reflect all the disease activity present in the airways that may affect the patient and spirometry results correlate poorly with other measures of health (see Section 3.1.1, p. 39).

Physicians appear to estimate their patients' health using criteria different from the patients themselves. Quality of life questionnaires provide a method of quantifying the effect of disease on patients' lives. They can summarize a number of aspects of the disease and provide an overall estimate of the effect of disease and benefits due to therapy. They have the potential to identify a threshold response to treatment that may be considered 'worthwhile', and allow comparison between therapies with respect to the health gain that each provides.²⁷² Therefore, for the purposes of balancing measurement of lung function with functional assessment a tool was required for this study, which can be used in research.

There are a number of questionnaires that are widely used in clinical research and attempts have been made to validate them and to compare one with another. Validation aims to assess whether a questionnaire measures what its authors claim but no single step or test will be sufficient to allow overall judgement.²³⁸

In general, questionnaires for respiratory health have two main properties. They can be used to compare and distinguish between the health status of different individuals (discriminative) or they may be used to detect changes in health over time or after treatment (evaluative).²³⁸

Essentially the SGRQ was a pragmatic choice for this research project. The instrument used needed to be able to discriminate between different levels of health between patients and to ensure that the distribution of respiratory health was more or less the same overall in both the control and intervention groups.

Changes (evaluative) in respiratory health after intervention were not measured and were not a primary or secondary outcome measure in this research project.

This tool has been used extensively in high profile studies such as the ISOLDE trial²⁷³ and continues to be used in new randomised controlled trials.^{236;238} Moreover it has been validated in many different settings and compared to other quality of life tools.²⁷⁴ The SGRQ has become a standard by which new questionnaires are judged²⁷⁵ and used extensively to assess the impact of treatments for asthma and COPD where the measurements such as FEV1 (Forced Expiratory Volume in 1 second) or PEF (peak expiratory flow rate) may not be significantly altered.^{276;277}

Other widely respected and used instruments such as the chronic respiratory disease questionnaire (CRQ)²⁷⁸, could have served the purpose equally well but the CRQ was designed specifically as an evaluative instrument and therefore would have been more useful if the study had been measuring changes in respiratory health as an outcome measure.

The SGRQ and the CRQ are too long to be used by most doctors in routine clinical practice. Symptom scoring tools such as the Medical Research Council dyspnoea score (see Box 1, p. 43) are brief and are useful in the clinical context because of their focus and ease of use but measure only one aspect of the impact of respiratory disease and they fail to capture the broader picture of a person's lung health on their quality of life. The Airways Questionnaire (AQ20) is a shortened version of the SGRQ and takes two or three minutes to

administer and was originally developed to assess asthma²⁷⁹ (Appendix 3, p. 292). The scores correlate moderately well with the SGRQ but there is poor correlation of scores with FEV1.^{279;280} Therefore the shortened forms could have been used in this study but are not as good as the full form SGRQ which may be regarded as the gold standard life impact measure.

In summary the SGRQ was chosen because it has both discriminative and evaluative properties²³⁸ and continues to be used in large randomised trials in intervention research in respiratory disease. Therefore, I describe the content and practical use of the SGRQ in detail in the next section.

5.6.1 Saint George's Respiratory Questionnaire (SGRQ).

The St George's respiratory questionnaire is a standardized self-completed questionnaire for measuring impaired health and perceived wellbeing ('quality of life') in airways disease. It has been designed to allow comparative measurements of health between patient populations and quantify changes in health following therapy.²⁸¹

The full questionnaire (see Appendix 3, p. 294) is a six page self-completed, supervised questionnaire. The participant is asked to make observations about their own health and in particular how their chest, cough and breathlessness influence their daily life. Three component scores are calculated: symptoms, activity, and impacts (on daily life), and a total score.²⁸² Guidelines are also available regarding completion and interpretation. The three component scores are explained in the guidance as follows:

Symptoms- this component is concerned with the effect of respiratory symptoms, their frequency and severity.

Activity- concerned with activities that cause or are limited by breathlessness

Impacts- covers a range of aspects concerned with social functioning and psychological disturbances resulting from airways disease.

A total score is also calculated which summarizes the impact of the disease on overall health status. Scores are expressed as a percentage of overall impairment where 100 represents worst possible health status and 0 indicates best possible health status. Normal values for normal subjects (FEV1 91-99% of expected value) have been calculated (total score 6 [95% Confidence Interval 5-7]) and the calculator will function with as much as 24% of data missing.

Studies have been done to correlate SGRQ scores and FEV1 in COPD and asthma (see Figure 7, p. 181). This plot was produced in 1999 and included in the manual for the SGRQ. Since then many studies have been published for asthma and COPD. In them, the mean values for FEV1 and the associated SGRQ scores lie on or close to this regression line. The authors state that a difference of four units between scores is clinically significant. This difference applies to a change within an individual over a period of time and between groups of patients.

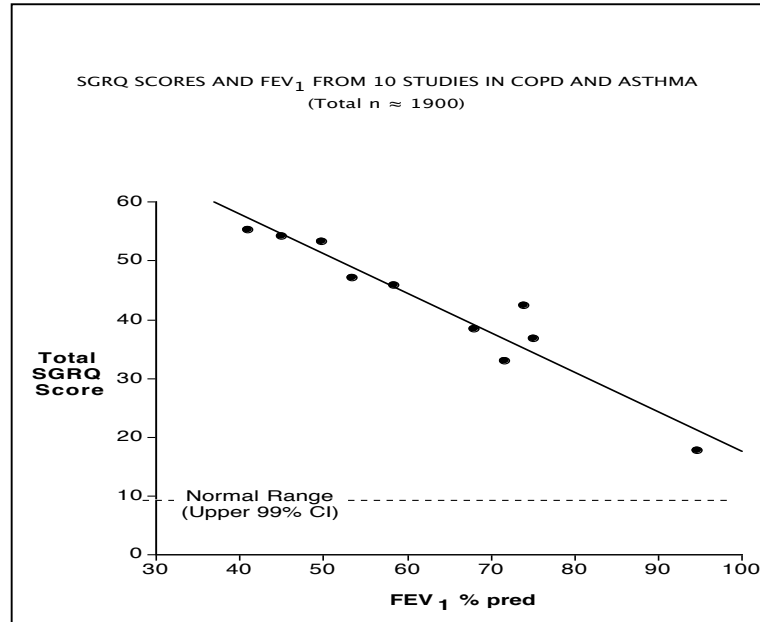


Figure 7 FEV₁ correlation with SGRQ scores

In the pilot study for this research project I found that most literate participants could complete the questionnaire with a small degree of supervision and clarification. Those with reading difficulties were able to respond to the questions when read out loud. Invariably before the participant departed it was necessary to ensure that they had not missed any sections but otherwise they had a high rate of successful completion and took no more than 20 minutes to complete.

6 Methods

6.1 Management and governance

6.1.1 Ethical approval and management

The study was approved by Hertfordshire Local Research Ethics Committee (application number EC03718) and West Essex Local Research Ethics Committee (1608-0104) and registered prospectively on the National Research Register (ID no. N0096173751).

I had established a practice based core management group to assist me with the day-to-day running of the project, which included the practice manager, two practice nurses, health care assistant, and a patient representative. The academic and clinical research advisory group was set up and consisted of a local consultant respiratory physician (RD), academic general practitioner (TG) and the statistician (MG) as well as myself as principal investigator (GP).

6.1.2 Sampling and recruitment

A randomised controlled trial was chosen as the best study design to determine the effect of the intervention whilst avoiding bias and reducing influence of confounding factors. We considered the possibility of contamination between participants. In practice this was not likely to be a problem as the intervention was given on an individual basis. Unless the participant (in the control group) went elsewhere to 'get their lung age measured' then there was minimal risk.²⁸³

Cluster randomisation was considered at the development stage as it would have simplified individual practice protocols but it would have required a larger numbers of participant (GP) practices than for individual randomisation. For example we would need to recruit a total number of 16 clusters (practices) and total sample size of 318 participants, assuming an intra-class correlation of 0.007 and a cluster size of 20 participants, inflated to take account of attrition.²⁸⁴⁻²⁸⁶

Therefore, individual randomisation was the design of choice with the sample size chosen to have 80% power to detect a 10% difference in smoking cessation rate (e.g. 5% in one group versus 15% in the other group). A power calculation indicated the need for approximately 300 participants (137 in each group). We chose a ten percent difference from the intervention as a useful clinical improvement, which would compare well with other interventions described in the literature. Assuming an attrition rate of up to 50% we aimed to recruit 600 participants.

With the cooperation and assistance of each of the five practice managers, I organised a search of computerized patient records from five general practices in Hertfordshire, and West Essex, to identify people aged 35 and over who had been recorded as a smoker during the previous 12 months. A full list of exclusions and exclusions can be seen in Box 9 on page 184. Those on oxygen therapy and those with a history of lung cancer, tuberculosis, asbestosis, silicosis, bronchiectasis or pneumonectomy were excluded. The rationale for excluding those with the diseases listed is that the lung age measurements from

those individuals cannot be a true reflection of lung damage due to smoking and therefore can not be a legitimate educational or motivational tool when explaining results to smokers. Those on oxygen or housebound were excluded due to the practical logistics of conducting the research and the likelihood that the severity of lung disease or co-morbidity would preclude spirometric testing or meaningful lung age assessment.

Box 9 READ codes for computer search

1. Currently registered	SHARED
2. Current smoker. 137R Date range 9.11.02-todays date	SHARED
3. Malignant Neoplasm bronchus B22z	EXCLUDED
4. Bronchiectasis H34	EXCLUDED
5. Asbestosis H41	EXCLUDED
6. Pulmonary Tuberculosis A11	EXCLUDED
7. Lobectomy of lung 74512	EXCLUDED
8. Age between 0Y and 34Y	EXCLUDED
9. Achondroplasia PG41	EXCLUDED
10. Silicosis H421 H422.	EXCLUDED
11. House-bound. 3CA	EXCLUDED
12. Oxygen concentrator. Anytime. 5061 EMIS.	EXCLUDED
13. Current Drugs, Oxygen cylinder in last 2 years	EXCLUDED
Also exclude:	
14. Unable to give consent e.g. severe senile dementia, severe psychiatric illness etc. (no EMIS code available)	

The ethics committees had approved the format of the invitation letters and the information sheet about the research. With the help and cooperation of each of the practices the invitations and information was sent to each of the people identified as current smokers on the registers. These were sent out on the practice headed notepaper in batches in order to spread the responses and follow up. Two weeks later, we telephoned all those who had not already responded, offering an invitation to participate and to answer any queries. Those who could not be contacted by telephone were sent a second letter. Posters were put up in the waiting rooms of each of the practices and clinicians in each of the primary care centres were asked to promote the study on an opportunistic basis and to encourage smokers to respond to the invitation to participate. Recruitment started in February 2004 and follow up was completed in March 2007.

6.2 *The assessment interview*

All potential participants were asked to confirm that they were current smokers, had understood the information provided, and would be available for re-assessment in 12 months time. Baseline data included age, smoking history in pack-years (average daily cigarettes divided by 20 and multiplied by the number of years smoking), medical history for exclusion criteria (see Box 9, p. 184), medication (especially use of steroids or antibiotics for chest infections in the preceding 12 months), and co-morbidity including chronic bronchitis or emphysema, asthma, other lung disease, diabetes, treatment for blood pressure, stroke, coronary heart disease (angina or heart attack) or other heart disease. These co-morbidities were not used as exclusion criteria but to

confirm baseline comparability of groups and to make the study as inclusive as possible and representative of real life primary care populations of smokers (see Section 8.2.1, p. 205).

All participants had standard lung function measurements (FEV₁, FVC, FEV₁/FVC) using a Micromedical® spirometer. Reversibility of airways obstruction was measured according to standard British Thoracic Society guidelines.⁴⁷ Both groups were told that their lung function would be measured again after twelve months to see whether it had deteriorated. At this stage they were not told whether they were in the intervention or control group. All participants were strongly encouraged to give up smoking and advised how to access local NHS smoking cessation clinics.

Two instruments were used to confirm baseline comparability of groups: the St George's Respiratory Questionnaire (SGRQ) (see Section 5.6.1, p. 179) and Prochaska's stages of change questions in relation to smoking. The SGRQ is a validated questionnaire designed to be self-administered under supervision and to measure the impact of respiratory diseases (in particular asthma and COPD) on an individual's life.²⁸¹ Like other quality of life instruments, it has the potential to identify a threshold response to therapy and/or compare the response to different therapies.²⁷² According to the authors scores of 7 or below are found in those with normal lung function. For full discussion of the choice of the SGRQ see Section 5.6.1 (p. 179).

Stage of Change questions were adapted with authors' permission from

Prochaska and DiClemente's model in which smokers are asked three questions and classified on the basis of their response as in the 'pre-contemplative', 'contemplative', 'preparation' or 'action' phase (see Figure 6, p. 120 and Box 3, p. 117) (see Appendix 4, p. 301).

6.3 *Randomisation procedure*

A clerk (who then took no further part in the study) had prepared 600 sequentially numbered opaque sealed envelopes. Each envelope contained a card with allocation group determined by computer generated random number (odd = intervention). Each card had the group (intervention or control) and written instructions for what information should be given to the participant. This was not opened until after the lung testing had been completed.

If the participant met the inclusion criteria and gave written, informed consent, he or she was entered into the study and baseline spirometry completed. After lung testing had been completed, the next numbered envelope in the series was then opened to determine allocation group. The written instructions about what group and what information to given was then followed.

6.4 *Instruments and tests*

I organised the training for all data collectors in the use of MicroLab 3500 (micromedical Spirometers²⁴¹) which were newly purchased at the start of the study. They were instructed to check the readings for internal reliability on three criteria: (a) at least two FEV₁ readings within 5% of each other; (b) the time

volume curve of good quality; and (c) the internal spirometer computer display had to register 'good blow'. They then measured carbon monoxide levels using Micromedical CO monitor.

All the results were sent to the central address for the research project. On a weekly basis I checked the new data for completeness and quality and completed the letters to participants and their general practitioner.

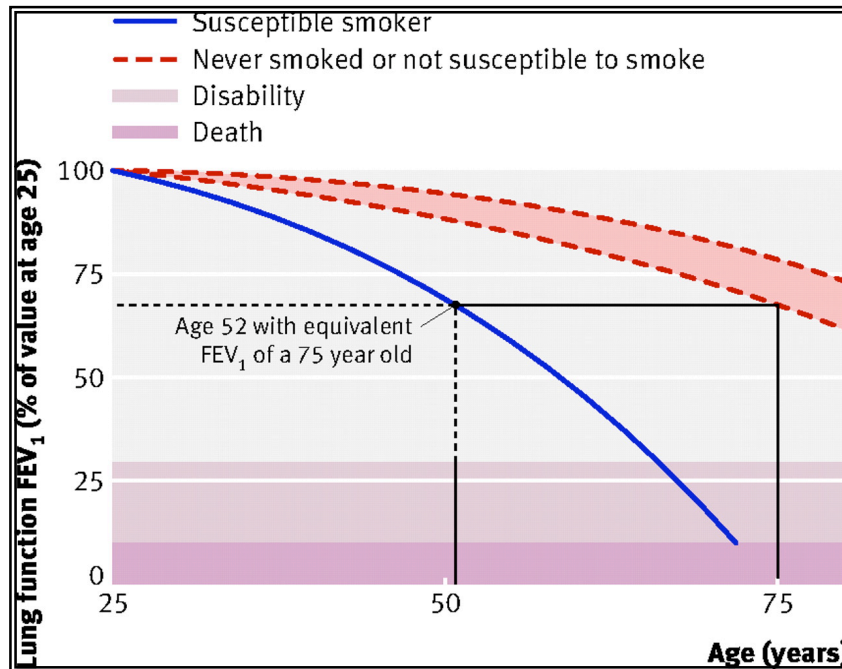
For the follow up testing, I recruited two independent nurses who were employed to contact each person who had made a self declaration that they had quit smoking at follow up examination. One of these two nurses then performed saliva sample collection from each participant in their homes. They were blinded to allocation group. They also recorded those who continued to take nicotine replacement therapy (NRT).

6.4.1 Lung age estimation

The concept of lung age estimation can be demonstrated graphically using a picture (Figure 8, p. 189), adapted from the work of Fletcher et al, which was used to show diagrammatically how smoking effectively 'ages' the lungs.^{6;6} The example in Figure 8 (not to scale) shows how a man of 57 with a FEV1 around 75% of the average value for his age has a 'lung age' of 77. The graph illustrates a much more complex calculation based on estimates developed by Morris et al using reference linear regression equations to establish the best method (see Section 3.1.1, p. 44).^{72;72} These authors showed that FEV1 was the best test for calculating lung age using a mathematical calculation (see Equation 1, p. 45). In practice, adjusting the settings of the spirometer

automatically generates the individual's lung age.

Figure 8 Lung age illustration adapted from Fletcher and Peto 1977



6.5 What participants were told about their lung function

Within four weeks of recruitment, data collection and testing, I sent each of the participants their results by post, in a personalised letter (see Appendix 5, p. 306). I first checked the quality of the spirometry tracing and considered the result in the light of clinical data. Where there was doubt, I sent the results to a chest physician (RD- clinical supervisor) for interpretation and advice (specialist interpretation was only required on six occasions). Written results were given to the control group as simple FEV₁ (litres per second) with no further explanation. The intervention group were sent their results as 'lung age' with a graphic display.

All participants were sent a standardised explanation of how smoking can damage their lungs. In the intervention group, if the lung age was equal to or less than the individual's chronological age, he or she was informed that their test result was normal, but smoking cessation was still recommended due to the risks of lung cancer, heart disease and stroke. If their lung age was greater than their chronological age, the 'lung age' was given in years.

In both the control and intervention group, when reversibility testing indicated asthma (over 15% and at least 400 ml improvement in FEV₁ after 400 mcg salbutamol via a spacer device), I sent a letter to the participant advising them to attend their general practitioner for further management, and a copy to their doctor. When spirometry findings suggested restrictive lung disease, I sent a letter to the participant advising them to attend their general practitioner and a letter to the general practitioner to alert them to the advisability of further investigation and guidelines on diagnosis, investigation and referral to secondary care (see Appendix 5, p. 308).

6.6 Outcome measures

The primary outcome measure was verified cessation of smoking twelve months after the initial recruitment interview and examination. Secondary outcomes were changes in daily consumption of cigarettes and the identification of new diagnoses.

6.7 Follow up and confirmation of cessation

Follow up examination with repeat spirometry was performed after twelve months. Self-reported quitters had carbon monoxide breath testing immediately for confirmation of smoking cessation. Within four weeks of attendance for follow up I reviewed the results of the spirometry and compared them with the initial test results. I sent each participant a letter, which compared their two tests and any change detected. For those who had reported that they had quit, I informed them in writing that they would be contacted by an independent nurse for a confirmatory test with a brief outline of what the salivary cotinine test would involve.

6.8 Data analysis

Data were analysed on an intention to treat basis. Statistical analysis was performed using SPSS version 11.0. Continuous data were analysed using the unpaired t-test. Categorical data were analysed using the chi-squared test, except where expected cells were found to be less than 5, in which case Fisher's Exact test was used.

To test the hypothesis that severity of lung damage predicts quit success, the t-test was used to compare the mean 'lung age deficit' (difference of lung age minus chronological age) between quitters and non- quitters, within the intervention group only.

6.9 *Assessment of costs*

This study was not designed to include a full economic evaluation. However, I had approximate data on the time taken for the spirometry tests to be carried out and for results to be communicated to patients by letter. Costs were calculated in terms of the professional and administrative time required per patient processed and also per successful quitter. The capital expenditure for equipment (spirometers and carbon monoxide monitors), start up, training costs and continue testing were not included in the estimates.

7 Results

7.1 *Baseline characteristics*

We recruited 561 participants, whose baseline characteristics are shown in Table 5 (p. 194). There were few statistically significant differences between the two groups at baseline; in particular, groups did not differ in their quality of life score (SGRQ) or stage of change. However, there were significantly more people with a history of stroke in the control group. The incidence of co-morbidity was high (around 20% of all participants), reflecting the deliberate intention of the study not to exclude high-risk individuals (and, perhaps, the inability or unwillingness of many smokers to quit despite the presence of significant medical morbidity).

Despite an average of 33 pack-years years of smoking, most participants in this study had 'normal' spirometry at baseline, which accords with previous studies on comparable populations.⁵ Using BTS cut-off values, only 23.5% of the control group and 26.8% of the intervention group had baseline lung function in the 'abnormal' range.

Table 5 Baseline characteristics of participants

	Control (n = 281)	Intervention (n = 280)
Age- mean (SD)	53 (11.9)	52.9 (11.9)
% male (n)	47% (132)	45%(127)
Pack-years -mean (SD)	30.3 (19.3)	31.1 (17.7)
Daily cigarette consumption -mean(SD)	17.4 (8.2)	16.5 (9)
Spirometry result		
FEV ₁ % predicted-mean(SD)	90 (19.8)	89 (19.8)
% FEV ₁ /FVC- mean (SD)	75 (11.8)	73 (11.7)
% with Abnormal FEV ₁ (n) (i.e. < 80% of predicted)	23.5 (66)	26.8 (75)
SGRQ score -mean (SD)	28.9 (22.4)	26.7 (22.0)
Past medical history % (n)		
COPD	7.2% (19)	7.7% (20)
Asthma	11% (29)	9.3% (24)
Other lung disease	2.7% (7)	2.3% (6)
CVA or stroke	4.2% (11)	0.8% (2)
Coronary heart disease (angina or heart attack)	5.3% (14)	2.3% (6)
Other heart disease	2.3% (6)	1.2% (3)
Diabetes	5.7% (15)	3.5% (9)
Treatment for hypertension	21.3% (56)	19.1% (49)
Precontemplative	29.3% (77)	29.2% (76)
Contemplative	32.3% (85)	31.9% (83)
Preparation	16% (42)	18.1% (47)
Action	22.4% (59)	20.8% (54)
New diagnosis of COPD	17.4% (49)	14.3% (40)
FEV1=forced expiratory volume in one second; FVC=forced vital capacity; SGRQ=St George's respiratory questionnaire; CVA=cerebrovascular accident; CHD=coronary heart disease; COPD=chronic obstructive pulmonary disease.		

7.2 Progress and outcome

Progress and losses to follow up are shown in the flow diagram below.

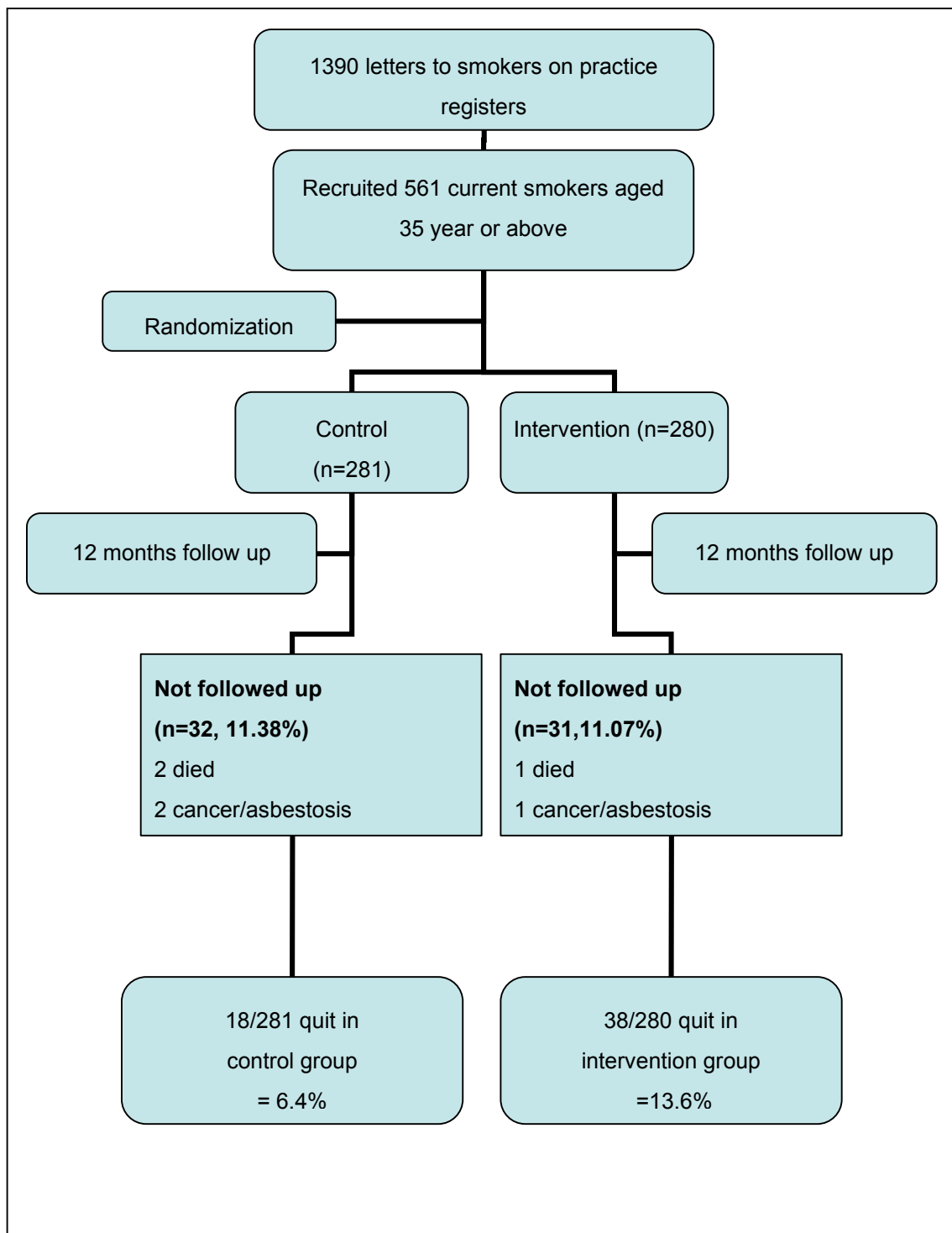


Figure 9 Progress and outcomes

Follow up data at 12 months are shown in Table 6 (p. 196). All recruited participants were included in the final data analysis. Those not returning for follow up (32 and 31 people respectively in the control and intervention group) were analysed as if they continued to smoke.

Table 6 Results at 12 months

	Control (n = 281)	Intervention (n = 280)	P value
Lost to follow up	11.4% (32)	11.0% (31)	0.9
Smoking status			
Confirmed cessation (cotinine CO)	6.4% (18)	13.6% (38)	0.01
Smoker at 12 months	90.4% (254)	84.6% (237)	
Unknown	3.2% (9)	1.8% (5)	
Self-reported cessation	15.2% (37)	20.2% (44)	
Average daily cigarettes	13.7 (SD 10.5)	11.7 (SD 9.7)	0.03
Attended NHS smoking clinics	1.4% (4)	1.7% (5)	
Used smoking cessation help (Clinic, NRT, bupropion , acupuncture)	7.8% (22)	10.7% (30)	0.2 (chi square test)
Quit rates in different stages of change ^k			
precontemplative	3.9% (3/77)	6.7% (5/75)	
contemplative	5.9% (5/85)	14.5% (12/83)	
preparation stage	11.9% (5/37)	14.9% (7/47)	
action stage	8.5% (5/59)	18.9% (10/53)	
not classified ^l	0% (0/18)	18.2% (4/22)	

All recruited participants were included in the final data analysis. Those not returning for follow up (32 and 31 people respectively in the control and

^k Exploratory data (not published in BMJ). Study not powered to give statistical significance

^l Pilot study of 40 participants did not include stage of change measurement

intervention group) were analysed as if they continued to smoke. Verified quit rates were 6.4% in the control group and 13.6% in the intervention group (difference = 7.2%, $p=0.005$, 95% CI: 2.2% to 12.1%). Telling participants their lung age was thus associated with an absolute reduction of 7.2% in the smoking rate compared to giving them their lung function tests results as raw FEV₁ data. The number needed to treat (NNT) for the intervention to achieve one additional sustained quitter is 14. Both the control group and the intervention groups reduced their average self-reported cigarette consumption; average consumption of cigarettes at follow up was significantly lower in the intervention group (11.7 per day, SD 9.7) compared to the control group (13.7 per day, SD 10.5) ($p=0.027$).

The average lung age deficit in quitters was 8.0 and in non-quitters was 8.8. The difference was not statistically significant. Thus there was no evidence to suggest that success at quitting is related to severity of lung damage.

Data for quit rates in the different stages of change in the control and intervention groups are included in Table 6. Statistics are not included for their significance as the study was not powered to detect differences between these subgroups. These figures reveal a trend to relatively better quit rates in the intervention group for all stages of change and in those in the stages which are said to measure 'readiness to change'.

7.3 Costs

The time taken for the health care assistant to undertake a spirometry test was approximately 30 minutes; GP spent a further 15 minutes per patient reviewing results and preparing a personalized feedback letter, and this required approximately ten minutes of secretarial and receptionist support. Using 2007 salary costs for the relevant staff, we estimate the cost of this intervention at £20 per patient processed and £280 [previous figure x 14] per successful quitter (given a NNT of 14). The economic burden of disease of COPD in the UK is estimated to be an average of £ 820 per annum.(£150 per annum for mild COPD)(2004)⁶⁷.

8 Discussion

8.1 *Summary of findings*

This study is the first published large RCT with adequate follow-up and independent proof of cessation to demonstrate that personalized 'lung age' feedback is effective in promoting smoking cessation.⁸⁴ This study strongly supports the policy of giving patients their spirometry results expressed as 'lung age' along with advice about the dangers of continuing to smoke and methods of quitting.

In 2001 a non-systematic overview analysed twelve studies that provided feedback on personal biomarkers as part of behaviour change strategies in smokers.¹⁴⁷ The authors concluded that success was likely to depend on how the information was conveyed and understood, and how it related to behaviour. They also suggested that success may depend on graphic displays or written personalized information as well as the prospect of gain rather than negative messages about costs or disadvantage.

A Cochrane review of the evidence for the effectiveness of biomarkers in smoking cessation was published in October 2005. Observational studies were included in the background discussion but only RCTs were included in the analysis, which concluded that due to limited evidence no definitive statements could be made about the effectiveness of biomarker assessment as an aid for smoking cessation.¹¹⁵ None of the primary studies included in the Cochrane review had used 'lung age' in the intervention. The negative conclusions of that

review should be updated in the light of this new study.

The debate about the usefulness of screening with spirometry was recently rekindled by a large non-randomised observational study of 4494 smokers from Poland.¹⁵⁷ Their results indicated that spirometry promoted cessation. Those with airways obstruction were more likely to quit, but even the group with normal lungs on spirometry had a higher quit rate (12.0%) than would normally be expected after simple physician advice (4-6%).¹²⁴ In this study, 'lung age' was not used to explain results to participants, but the authors did use a visual display of Fletcher and Peto's diagram⁶ (Figure 1, p. 44) to compare the participant's result with average-for-age and project the likely deterioration with continued smoking. These authors did not have a control group but attributed the high quit rates in those with normal lung function to a 'healthy volunteer' effect (those who had opted for the programme were seen as more motivated to quit).

The results of our study are broadly consistent with the findings of the Polish study, with one important difference. Contrary to the conclusions of the latter (and to clinical speculation), we demonstrated that successful quitting is not dependent on the severity of lung damage as demonstrated by spirometry. A 45-year-old smoker who is told that their 'lung age' is normal is as likely to quit as one who is told that his or her 'lung age' is 65. Giving information in an understandable and visual way, whether the news is positive or negative, seems to encourage higher levels of successful smoking cessation than those who are given feedback that is not easily understandable.

This begs the question of what triggers the decision to quit, and the mechanism by which successful and sustained quitting occurs. Clinical experience suggests that deterioration in health does not necessarily lead to altered behaviour whether that is smoking, drugs, or dietary. The high rate of co-morbidity (20%) in our sample population confirms that many who are likely to exacerbate a chronic health problem by smoking continue to do so. Anecdotally, some participants in our trial were relieved when the results were found to be normal and therefore felt it was 'not too late' to be trying to quit.

This apparent win-win situation may explain the apparently paradoxical finding that knowing one's 'lung age' helps a smoker to quit whatever the result. If 'lung age' is 'normal' there is an incentive to stop before it is too late. If 'lung age' is abnormal then this is a clear message that the lungs are undergoing accelerated deterioration, which would be slowed if the smoker quit (Figure 1, p. 44 and Figure 8, p. 189). Further research is needed to elucidate the psychological forces that are active in successful quitting in different circumstances.

Some addiction experts have proposed that the transtheoretical model should be rejected in favour of a new integrated model.^{92;231} Any new psychological theory of smoking cessation will need to explain the unexpected finding that normal results within personal biomarkers are as likely to promote cessation as abnormal ones.

In this study, we measured stage of change (using Prochaska and di Clemente's transtheoretical model) to ensure that the intervention and control groups were comparable for this variable at baseline, but the study was underpowered to test the hypothesis that a smoker in the 'active' phase of quitting would find lung age feedback more useful than someone in the 'pre-contemplative' phase. Nevertheless, the quit rates (Table 6) at the different stages reveal a trend which is supportive of the 'stages of change model'. The intervention, of using lung age as the motivating trigger, appears to work better at every 'stage'. Even quit rates for those in the precontemplative stage (6.7%) are better than the overall quit rate in the control group (6.4%). This data supports the notion that the intervention should be used in a non-targeted fashion towards the whole population of smokers rather than restricting it to those who declare themselves intending to change. This is in stark contrast to many other interventions including the NHS stop smoking clinics which target those in the preparation and action stage.

Current NICE guidelines include one on brief interventions and referral for smoking cessation^{29;287} (which do not mention spirometry testing at all) and another on the management of chronic obstructive pulmonary disease.⁶⁷ The implication is that spirometry testing is useful only when the patient has (or is suspected of having) established lung damage. The findings of this study suggest that both these guidelines should be reviewed and that 'lung age' testing (which is a quick, office based test that can be undertaken by a health care assistant) should be considered as part of a brief intervention package –

either in all smokers over 35 (the lower age limit for this study) or all smokers. Although this is a relatively brief intervention compared to NRT or motivational interviewing, it is a complex intervention as defined by the Medical Research Council guidance.²⁸⁸ The essence of a complex intervention is that it contains a number of interacting components and it is not certain which of the components is essential to the success of the intervention. The lung age intervention is at the simpler end of the scale of interventions to which the term applies. Caution is required about applying the lung age intervention to other groups or subgroups of smokers and in other contexts. This has importance when it comes to implementing the findings of the lung age study.

Currently the New Contract for General Practitioners in the UK (new GMS, Quality and Outcomes Framework) includes incentives to confirm the diagnosis of COPD using spirometry and to record smoking status in those with a record of significant co-morbidity (Coronary heart disease, hypertension, diabetes, CVA and asthma) and to give cessation advice. However there is no incentive to actively case find COPD among smokers (or ex-smokers) in these high-risk groups or in the general population. We therefore recommend that the new NHS GP contract (Quality and Outcomes Framework) should include incentives for spirometric assessment accompanied by personalized communication of 'lung age' in smokers. However great care needs to be exercised before suggesting that doing spirometry and giving the result as lung age will be as effective as the whole of the (complex) intervention including explanation of the graphic display and written personalised results. Preserving the fidelity of the intervention may require a more robust vehicle for implementation such as

through Primary Care Trust funding (e.g. local enhanced services).

Our cost estimates, which assume that spirometry is carried out in UK general practice, suggest that 'lung age' estimation and communication is of comparable effectiveness to, and potentially cheaper than, other currently available treatments on the NHS including nicotine replacement therapy (NRT)¹²², bupropion¹¹⁴, face-to-face counseling¹²¹, and telephone counselling.²⁸⁹ Given the heavy health and economic burden of smoking, we believe that formal economic evaluation of this new and simple intervention should be a research priority.

8.2 *Strengths and weaknesses of the study*

With the benefit of hindsight I have considered that a number of changes would have improved the design and conduct of this study. The following observations and comments are based on the benefit of ideas about things which I believe could have been done differently. Where there are good reasons that I did not do them I discuss these. Where there were areas of omission I will try to explain why they would have been beneficial.

8.2.1 Strengths of the study population

In this section I will show how the study gains credibility from being as inclusive as possible of those from the general primary care population and reducing the exclusions to a minimum as described in the methods section (see Section 6.1.2, p. 182).

Inclusion and exclusion criteria

Many studies exclude those with any significant co-morbidity, including known COPD or even those who had had spirometry in the previous twelve months. As this is the first study to use the concept of lung age I believed it was important and reasonable to include those people as they would have a 50:50 chance (by randomisation) of receiving their results in the new format (in terms of lung age). Furthermore, I wanted the population of smokers to be representative as possible of those encountered in real-life general practice. Those smokers with co-morbidities of diabetes, coronary heart disease, and hypertension etc. have usually been told many times of the increased risks with continued smoking. Therefore, despite the fact that this group includes those who are very resistant to advice and change, it is important to test any new intervention on those who

could benefit most from quitting.

Many other studies of smoking cessation interventions exclude those who have poor motivation to change their behaviour as judged by one of several different possible questions (e.g. the stages of change algorithm placing them in the pre-contemplative and contemplative stages).¹⁵⁹ The usual reason given for excluding those not intending to quit is that they will not be helped as much as those who are ready for change and that interventions should be targeted towards those who will benefit most. I had no reason to believe that the intervention would be better for those who were ready to change and therefore did not exclude those who are self-declared not contemplating quitting and are therefore thought to be resistant to change.

The study was not promoted as a programme for quitting smoking per se. I deliberately wanted to explore whether those resistant to change would respond to new information about their health. Although lung age might be regarded as a frightening tool or a scare tactic I prefer to think of it as giving personal-health information in an understandable and meaningful way. I hoped that communication of the information would allow people to make informed decisions. Because of the inclusive nature of the recruitment policy it is anticipated that the results are more likely to be transferable and generalizable to other general practice or primary care populations.

8.2.2 Weaknesses in data collection

Depression score

On a similar theme, I did not exclude those with mental health disorders except under the general exclusion of those unable to understand the information sheet or give informed consent. This would not exclude most people with depression. Participants were not asked to complete any type of depression questionnaire. In hindsight there are several good reasons that it would have been good to have a baseline measure of depression. For example this would have been a useful co-morbidity to list as those with depression are at risk of being neglected by smoking cessation campaigns and interventions and they are a vulnerable group with high rates of relapse after cessation.²⁹⁰ Furthermore it could have been useful to gather data about any association of knowledge of lung age and changes in depression scores. This would have been extra evidence of the ethics and safety of this intervention in different subgroups. Conversely this had not been considered as a primary or secondary outcome and therefore including an additional depression questionnaire would have added extra time and complexity to the study. Other issues about the meaning and impact of giving bad news (poor lung age) to depressed or anxious people or those with a history of depression is an area of concern as the information has the theoretical potential to cause relapse.²⁹¹ This will be discussed in more detail in the section on ideas for further research.

Measure of Nicotine dependence

On reflection I could have included a score of nicotine dependence in the initial data collection. This would have strengthened the evidence that the control and intervention groups were alike and also been a valuable additional dimension

and add integrity to the study. However, because this was a randomised controlled trial the findings remain valid even if the degree to which nicotine dependence played a role is not known. The fact that very few people in either group admitted to using smoking cessation clinics or nicotine replacement therapy highlight the possibility that this study is complementary to use of NRT rather than in direct competition with that intervention.

Social and economic status

Some of the feedback has cast doubt on the generalisability of the results to other populations. It would have been useful to collect socio- economic data on these patients.

Smoking history of the family

There is clear evidence from other studies that living and socialising with smokers is a barrier to continued abstinence. Secondly, there is also evidence that false positive exhaled carbon monoxide can occur with passive smoking. Data about the smoking habits of the household would have helped in interpreting some of the results but overall would have had little effect as the primary outcome measure was cotinine levels in saliva which is unlikely to produce false positive results with passive smoking.

Body mass index

Some of the normal scales for respiratory function include the patients weight as this is know in some circumstances to effect the results of lung function tests. The reference tables for FEV1 that I used in this study do not require entry of this data but weight was probably relevant in a couple of cases where extreme obesity affected the FEV1. It could have been useful and very little extra work and time would have been necessary to collect the extra data.

Self- efficacy

Various psychological theories have been discussed with reference to behaviour change. It is important to emphasise that this research study was not testing a particular behaviour theory. There were no preconceived ideas about how the intervention would work. Rather, the study was designed to test if the intervention worked rather than how it might work. In hindsight the concepts of self-efficacy in particular are very interesting and possibly have a role in mediating the changes that were seen in this study. Although it would have been interesting this was not the main aim of the study and data collection would have become too complex and time-consuming if all these different aspects had been covered. It is also possible that the process of collecting that sort of data, in a sense, becomes part of the intervention – as the types of questions trigger a series of thoughts attributing change to ideas that might not otherwise be present.¹⁶² In conclusion I believe that it was correct not to divert the research into chasing a theory prior to demonstrating the efficacy of the intervention. However this has already acted as a stimulus to new research^{56;270} and comment and I include a few ideas for further research (see Section 8.3, p. 210).

Definitions of abstinence

The nature of this research was that the intervention was a one-off measurement and information-giving exercise with written personalised advice but without lengthy counselling or other measures to promote smoking cessation. Point prevalence with biochemical validation was the logical choice of measuring smoking status and twelve months was chosen as the best period in keeping with other research and the period for which the study was funded. In

hindsight I could have measured three month or six-month progress rates, as I did at the pilot stage, but in reality the final end-point of interest was the status after twelve months. I could also have measured quit attempts but these are surrogate end-points, which do not necessarily lead to lasting change. An important next step is to do long term follow up and retrospective analysis of the reasons that the smokers quit in this study.

8.3 Further research

A number of researchers have tried to use biomedical markers to motivate change in behaviour. The most recent studies have advanced knowledge about what might work with regard to giving information to smokers about the health of their lungs.^{84;157} Many authorities will neither fully accept nor incorporate the results into clinical practice or policy until they have been replicated. Therefore it is assumed that publication of further studies of lung age and its effect on smokers would add value to this study. In particular it would be useful to know how well the intervention works in different subgroups of smokers depending on age, ethnic group or socio-economic status.

This research and thesis has uncovered some surprising results. In particular the results have produced some broad questions about the use of lung age and the mechanisms of action of the intervention. For example:

- What do smokers and non-smokers understand by the term lung age?
- What are the different psychological forces involved in quitting when smokers are presented with their lung age?
- Are these forces influenced by mental illness especially depression?

- What is the effect of receiving bad news about lung age on psychological health (e.g. anxiety, depression)?
- What is the value in early diagnosis of COPD?
- Does screening for early COPD result in better outcomes e.g. reduced progression, earlier smoking cessation, less mortality?
- Is there value in targeting lung age assessment according to other criteria such as stage of change or nicotine dependence?

8.3.1 Qualitative research questions

The original concept developed by Morris and Temple⁷² and this lung age study have provoked some interesting questions about the meaning of lung age. Of particular interest is the meaning that smokers may attribute to the information given. Intuitively, it would have been easier to understand or attribute meaning to the concept of lung age, if those with the worse results (highest lung age to real age deficit) were the most likely to be motivated to quit smoking. That concept was certainly considered in previous papers about the improved quit rates in those with lung damage in non-randomised trials.¹⁵⁷ However, the fact that the lung age intervention was associated with better quit-rates irrespective of the result begs many new questions.

In the discussion of the results in this thesis (see Section 8.1, p. 199) I considered why good results might lead to better quitting and why bad results might not, and vice versa. I suggested a win-win situation where those with good results are more motivated to quit because 'it is not too late to change' and those with poor lung age are motivated by the idea of halting progression of damage.

Using complex models from cognitive theories I might be able to theorise about what cognitions are active in different types of people and this will be discussed in the next section. However from a qualitative point of view I think there would be great value in studying what smokers understand by the concept of lung age and furthermore studying different 'new typologies' of smoker that have emerged from the lung age study as follows:

1. Normal lung age and failure to quit smoking
2. Normal lung age and successful quitting
3. Abnormal lung age and failure to quit
4. Abnormal lung age and successful quitting

It could be of great value to gain an understanding of the range of meaning that is attributed to their results by these different groups. Within each group it may be that there is also a range of meaning, which triggers a variety of responses, including continued smoking, attempts at cessation or successful cessation.

Possible methods

Focus groups

Initially it would be valuable to arrange some focus groups of smokers (not previously involved in the research) to establish what range of understanding there can be with the concept of lung age. They could be presented with a number of scenarios of smokers with different results and an explanation similar to that given in the lung age study using a graphic of lungs aging and the possible effects of smoking.

Sample questions:

1. If your lungs cannot return to normal how much value do you see in stopping smoking?
2. If you found out that your lungs were functioning as if they are 10 years older than your age, how would you react?
3. If you found out that your lungs were functioning as if they are 20/30/40 years older than your age, how would you react?
4. How would you understand a lung age of 110?

Structured interviews

Having established a range of responses it would be possible to conduct structured interviews with people who had been in the lung age study to establish how they actually stopped smoking and whether the intervention had influenced them at all. The study would have to start from the neutral position of not assuming any one influence had been greater than another.

Possible open questions for ex-smokers from the lung age study:

- I understand that you used to be a smoker and have now quit.
5. What were the main things that kept you smoking for xx years?
 6. What did you like about smoking?
 7. What did you not like about smoking?
 8. What do you think were the factors that helped you to quit?
 9. How did you finally quit?

If the lung age study has not featured during the above open questions then proceed as follows:

10. What do you remember about taking part in the research study on smoking?
11. What happened at your first interview and examination?
12. What was your result and what did you think of the results?
13. What if any effect did that have on your desire to stop smoking?

Possible questions for continuing smokers from the lung age study:

14. What were the main things that have kept you smoking for xx years?
15. What do you not like about smoking?
16. What do you like about smoking?
17. Have you tried to quit smoking?
18. What do you think are the things that might help you to quit?
19. If you have never considered or tried to quit smoking can you think of any situation which might make you change?

If the lung age study has not featured during the above open questions then proceed as follows:

20. What do you remember about taking part in the research study on smoking?
21. What happened at your first interview and examination?
22. What was your result and what did you think of the results?
23. What, if any, effect did that have on your desire to stop smoking?

The above questions and the focus groups could be used in two ways. First the transcribed data would be analysed and coded for emerging themes.²⁹² Subsequent interviews should be coded with the earlier interviews in mind in order to compare data. A theme or variable may emerge allowing categories to be discovered. The process continues until no new themes are apparent (saturation). From this process it is hoped that a theoretical framework would arise. Secondly, once the range of possible answers has been explored, this data can be used to inform the development of a questionnaire. In this way by triangulation one might be able to understand more fully the possible effects of lung age in different circumstances. A questionnaire could then be piloted and then used on all the people who took part in the original study. Furthermore, as the qualitative data produces a better understanding it may be possible to develop more specific questions to be used in the prospective trials described below.

8.3.2 Follow up data on original study

Given adequate funding and time the first priority in the quantitative area of research would be to follow up those involved in the original study to discover the continuous abstinence and point prevalence smoking cessation rates two and three years after the intervention. This would be briefer than the one year follow up because spirometry assessment would be unnecessary. Ethical permission and new consent would be required. I would propose that all those who gave consent to be contacted in the future be contacted via the persons own GP after they check the personal details and state of health in order to avoid distressing mistakes or breaches of confidentiality.

8.3.3 Asymptomatic airways obstruction

There has been considerable debate about the group of people with asymptomatic lung damage that are emerging from opportunistic spirometry and that would arise from screening if this was implemented. As discussed elsewhere it is unknown whether there are differences between them and their counterparts who present with symptoms. Most of those with so-called asymptomatic airways obstruction have been detected in primary care and are not then retested or confirmed by 'experts' in respiratory laboratories or secondary care. There is continuing controversy about the accuracy of those results and value of detecting these individuals. Likewise there will be even more controversy when new technology allows individuals to test themselves. This begs lots of questions about accuracy and safety issues and the development of devices for self-diagnosis and self-monitoring.

Every clinician will recognise the situation where a patient denies symptoms but on simple clinical observation they are clearly breathless or wheezing after slight exertion. This denial is often couched in terms of 'I thought I was just unfit', or 'I have put on weight '. We do need innovative and objective ways of assessing primary care patients, which are quick and accurate and do not just rely on spirometry or symptom scores.

Studies should also assess what proportion of patients with previously undiagnosed airflow obstruction who present with a first COPD exacerbation does not receive a clinical diagnosis of COPD.²⁹³

There were nine cases of moderate or severe COPD discovered during the period of the lung age trial. It seems remarkable that these people had not noticed symptoms or consulted with a health professional. In our initial data collection we relied on self-reporting of pre-existing diseases used in the morbidity data. It may be that communication failed or there was lack of understanding of terminology and their disease was on their medical record. Unconnected with this research, I have personal experience of visiting a patient at their home while they breathed oxygen through a mask, and simultaneously denied knowledge of her COPD to a specialist respiratory nurse. Therefore there may be various reasons that they denied a pre-existing condition that was already on record and therefore this aspect of the research may have been an overestimate of new diagnoses.

8.3.4 Psychological and cognitive factors

The lung age study was designed to test the hypothesis that this complex intervention would have an effect on smoking behaviour but was not specifically designed to test mechanisms or to test a particular psychological theory of behaviour change. Nor was the study designed to target any one theoretical determinant of behaviour change.

The best way to test the theory, that the effect of using lung age is associated with or mediated through specific cognitive factors to produce changes in behaviour, is to do a prospective trial and include an initial assessment of those variables of cognition. The most practical method would be to ask questions designed to test variables thought to be active such as self-efficacy, threat aversion or perceived susceptibility depending on the theory being investigated

(see Section 4.1.4, p. 96 and also Section 5.3.1, p. 159). Alongside those measures the study would also test factors that might interfere or modify the response for example nicotine dependency, culture, education and depression.

8.4 *Practice based research*

In this brief section I reflect on the process of primary care practice based research. This is not intended to be a comprehensive assessment of the past and future of research in primary care but some thoughts on how this fits in the context of caring for patients and dealing with other demands made on the primary health care team. I will also ponder the benefits to individuals, the team and the local community of primary care providers.

The Government is keen that research capacity should develop within primary care. The most recent statement in the new national health research strategy document declares the intention to 'create a National School for Primary Care Research'.²⁹⁴ Historically, research has been less well supported and funded in primary care compared to secondary care. The demise of primary care research was even contemplated by an editorial in *The Lancet* in 2004.²⁹⁵ A large proportion of health care activity goes on in primary care and good quality evidence for many activities is seriously lacking.²⁹⁶ It makes logical sense that interventions should be tested where they are used rather than extrapolating from highly selected secondary care populations.

There is probably no fundamental difference in the make up of practices which are involved in research and those which are not.²⁹⁷ Practice based research is

a process, which has been shown to develop in a non-linear way usually starting by the initial interest and enthusiasm of a single professional.²⁹⁸ Initially, any research activity may be seen by other members of the primary care team as a distraction from patient care and the business of general practice and an unnecessary use of time and resources. It may also cause conflict due to different priorities. However, if these barriers are overcome and the right funding and infrastructure and training are available the practice may move forward through a series of phases, which have been called transformation, consolidation, and moving on to collaboration.²⁹⁸ This transformed organisation will have a new culture and infrastructure to support research.

I believe that, as a practice, we have reached a critical moment where we could build on the success of the research venture and seek to become a 'research ready practice' with enthusiasm and infrastructure to support innovation and collaboration. We have applied to be included in the Primary Care Research Network (East of England) strategy for establishing a network of research practices.

8.5 *How should practice and policy change?*

How much change to practice and policy should come from the results of one study is a difficult question to answer. As the results are disseminated, and policy makers either accept or reject the findings, the amount of impact and what sort of change will happen as a result of this research will gradually unfold. Whether change should come about through gradual dissemination from the grass roots or whether change is better coordinated and managed from a top down process is a debate that is beyond the scope of this thesis. However, it is feasible that both may happen to some degree. The grass roots change can happen locally and in a small-scale way that is more manageable and can act as a beacon for others to follow when the practicalities have been ironed out.

After finding that the lung age concept influenced decisions by individual smokers we decided to use the information to try a practical demonstration of what a lung screening service might look like. We managed to secure six months funding from a pharmaceutical company to employ a respiratory nurse to invite and test all those on our practice register who were recorded as smokers or ex-smokers.

The aims of the programme were two-fold. First we were trying to detect more cases of COPD in our practice population as prevalence figures suggested that we continued to have a large number of undetected cases. Secondly, we were committed to use the research findings in a practical way to encourage more of the continuing smokers to quit. The first aim required that we test both current

and ex smokers and the second aim concentrated on those continuing to smoke.

A computer search of our practice database of 10300 registered patients showed our practice prevalence of COPD patients to be 1.62%. A further search revealed 2578 smokers or ex-smokers. We excluded those who are already on the COPD and asthma registers, those with lung cancer and asbestosis and those who had been in the intervention group of the lung age study. We included those over 35 year of age who had a computer record of being a smoker or ex-smoker.

During the 6 months of nurse funding, 305 smokers or ex-smokers attended for testing by spirometry. The nurse filled out a computer template of data for each of them, which included current daily consumption of cigarettes, smoking history to calculate pack-years, and symptoms (cough, wheeze and shortness of breath). Spirometry was done with the same equipment as in the lung age study with post-bronchodilator measurement where indicated.

Ex-smokers who attended had all data collected and were told about their results in a usual traditional way of having 'normal results' or a diagnosis of mild, moderate or severe COPD, according to BTS guidance. Those who had normal lung function were reassured. All those with any degree of COPD were offered annual flu immunisation and pneumonia vaccination and asked to attend early if they developed respiratory symptoms. Those with moderate or severe COPD were offered an appointment with a doctor to discuss the diagnosis and

treatment options.

Current smokers were given their results as lung age and all of them were offered help with smoking cessation through local clinics. If the FEV1 was under 80% and their FEV1/FVC ration was under 70 % then they were placed on the COPD register and given the same options as above for the ex-smokers for follow up.

From a practice perspective the programme was easy to run but the communication and relationship with the pharmaceutical company lead us to decide that we would not involve them again for a similar programme. Their decision-making was slow and cumbersome, they changed the point of communication three times during the run up to and during the programme and they were late paying us for the work done (the last payment came 4 months after the end of the pilot programme despite repeated promises of earlier payment). They also started requiring unprecedented access to our practice nurses and gave her false promises about courses and funding.

The preliminary data is encouraging. Just over 300 patients came for testing. Of those tested 17% (53/305) had previously unrecorded abnormalities. The majority had mild COPD (44), eight had moderate COPD and one was severe by BTS criteria.

It would be wrong to extrapolate the figures from this small pilot programme to justify a nationwide study but the costs are roughly 110 pounds for each new

case of COPD and 650 pounds to discover a new case of moderate or severe COPD, which would warrant medical therapy and preventative measures.

It is too early to say whether a twelve-month audit of the screening programme will show any improvement in follow up, influenza immunisation uptake or smoking cessation among those with smoking related lung disease. However, we have shown that a mini-screening programme is both practical and acceptable to our practice population although there is still work to be done to attract those eligible individuals who could not or chose not to attend the clinics.

8.6 Commercial impact

8.6.1 Health Enterprise East

Health Enterprise East is an NHS funded^m regional operation established to encourage new ideas to gain a commercial foundation and to enable innovations to be developed to maximise benefits for patients, NHS staff and the wider health community. Health Enterprise East (HEE) is the NHS Innovations Hub for the East of England, set up to help NHS staff across the six counties (Bedfordshire, Cambridgeshire, Essex, Hertfordshire, Norfolk & Suffolk) develop and take forward new innovative ideas – both products and services – to enhance healthcare delivery.

I have had some discussion with Health Enterprise East about the possible

^m HEE is funded by the Department of Health, Department of Innovation, Universities & Skills and the East of England Development Agency (EEDA).

expansion of availability of lung age testing by developing an information and instruction pack to implement a programme by Primary Care Trusts or other smoking cessation providers. A commercial organisation has been developing the technology to test flow through a small disposable tube using blue tooth technology for transmitting the results to a mobile phone. This has the potential to make lung age testing widely available to individuals without reference to a health professional. Although this is an exciting development and a tribute to modern technology it is a dangerous precedent to isolate one aspect of the research results and extrapolate that the same benefit would apply in other circumstances. It is not clear which parts of the intervention are indispensable to achieve the same quit rates as the lung age study. A hand held disposable measuring device could give a lung age but in isolation from advice to quit and without verbal and written advice as follow up. Quality control would be more difficult. How would the manufacturers ensure that those with odd results or deteriorating results were reliably informed of the need to consult a qualified clinician? These discussions are at an early stage but they offer the potential for disseminating the methods and techniques used in the lung age trial.

8.7 Government policy

8.7.1 Changes to Quality and outcome framework (QOF)

There have been two revisions of the indicators used in the Quality and outcome framework (QOF) since it was initially negotiated in 2003 and implemented in 2004. With any dynamic process it can be difficult to match the research evidence with practical application and inclusion in a framework like the QOF. Within the QOF, the emphasis on COPD and smoking remains high despite so many other competing areas of clinical need.

I welcome the most recent revision of QOF, which permitted inclusion of those with early changes of COPD into the diagnostic criteria. The initial inclusion criteria of COPD in the QOF were different from all other well-established guidance. The official rationale was that those with a percent predicted FEV1 greater than 70% of normal were unlikely to have symptoms and that emphasis should be on those with more severe disease and therefore there was no need to include these in prevalence figures or follow up or smoking intervention. In my opinion, there are several good reasons why this was misguided. It is clear that the best way to prevent disabling, symptomatic COPD is through smoking cessation. By excluding those with minor changes on spirometry they were reducing the potential contact with professionals during the period that the most good could be done. Admittedly at that time there was no good evidence that early detection of mild COPD would lead to improvements in rates of smoking cessation but there already was well established evidence that simple brief

advice from a health professional can be effective in reducing smoking. Dismissing asymptomatic or early obstructive changes as trivial could reduce the impact of any advice to quit. Furthermore, to initially choose inclusion criteria, which were different to every other national and international guideline, seemed to defy any type of logic. Not only was it illogical and unhelpful but it was not compatible with diagnostic criteria embedded in clinical computer systems or evidence based clinical guidelines.³⁹

Although important interventions such as long term oxygen therapy and pulmonary rehabilitation are not included as distinct entities in the QOF the rationale given by the contract guidance for the requirement for documentary evidence of annual monitoring, of the FEV1 of patients with COPD, is that deterioration can be detected so that treatment can be changed or referral made. The regular recording of FEV1 is therefore used as a surrogate for quality of care in these patients. Further discussion of this aspect of the QOF is beyond the scope of this thesis.

The results of this thesis should be a valuable contribution to the knowledge base for decisions about inclusion of indicators for the QOF. The new finding that knowledge of lung age helps improve smoking cessation rates does not necessarily support the notion that those with early asymptomatic airways obstruction should be included in the disease register as it is not known whether regular or repeated lung age testing has any further significant effect on smoking cessation rates. However the key to systematic lung age testing of smokers is to have good data collection and an accurate register of smokers.

It is well recognised that COPD is under-diagnosed and will progress with continued smoking.^{5;59;60;299} It therefore makes sense for those who smoke to have regular spirometry so that they may have ongoing advice and treatment for symptoms. Some of the counter arguments will be included in the next section about screening.

8.7.2 UK National Screening Committee (NSC) criteria

The National Institute of Health and Clinical Excellence (NICE) guidelines do not recommend spirometric screening of all smokers or ex- smokers for obstructive changes. Recently the U.S. Preventive Services Task Force (USPSTF) confirmed their previous advice that screening for COPD is not recommended.^{300;301 302}

In this section I review some of the criteria necessary for a test to be included in national screening programme and to what extent the current state of knowledge about COPD is consistent with those criteria.

The UK National Screening Committee (NSC) has published criteria for considering a test to be valid effective and appropriate as a screening test.³⁰³

The criteria are divided into four sections:

- The condition,
- The test,
- The treatment and
- The screening programme.

Ideally all the criteria should be met before screening for a condition is initiated.

The condition³⁰³

According to NSC

1. 'The condition should be an important health problem.'
2. 'The epidemiology and natural history of the condition, including development from latent to declared disease, should be adequately understood and there should be a detectable risk factor, disease marker, latent period or early symptomatic stage.'
3. 'All the cost-effective primary prevention interventions should have been implemented as far as practicable.'

There is overwhelming evidence that COPD is a very serious and growing health problem and there is a period in early disease where it is detectable before it has seriously damaged health. There is also clear evidence that there are a large number of cases that remain undiagnosed (see Section 3.1, p. 36). The main cause of COPD (in 90% of cases) is smoking and the only intervention, which will prevent progress of the condition, is cessation of smoking. There are now lots of cost effective interventions for helping people to stop smoking if they are willing to engage with cessation programmes.

As far as cost effective primary prevention interventions (criterion 3) the most effective methods to prevent damage are to reduce the numbers of young people starting to smoke. However, that discussion is beyond the scope of this thesis.

The test³⁰³

According to NSC

1. 'There should be a simple, safe, precise and validated screening test.'
2. 'The distribution of test values in the target population should be known and a suitable cut-off level defined and agreed.'
3. 'The test should be acceptable to the population.'
4. 'There should be an agreed policy on the further diagnostic investigation of individuals with a positive test result and on the choices available to those individuals.'

As already explained in detail the main test for COPD is spirometry, which is simple, safe, available in primary care and most patients are able to successfully perform the manoeuvres necessary. On the negative side there has been considerable disagreement between researchers, specialists and primary care workers about the quality, reliability and interpretation of spirometry done in primary care.³⁰⁴ Some studies have shown very high quality testing and interpretation of results and others have seriously failed to deliver quality.^{304;305} The USPSTF has called for more studies on the diagnostic accuracy of spirometry in primary care compared with the secondary care setting.³⁰⁰ There are two separate issues at stake in the argument. The first is about training the clinical or technical staff to perform the test with a sufficient skill and good quality control. The second is about knowledge of interpreting the graphs and figures.³⁰⁴ Any screening programme would need good training in both these areas and sufficient continuous monitoring to maintain quality. Any

problems could be explored during a pilot project for the screening programme before this is rolled out to the whole country.

In addition there is plenty of discussion about the differences between the diagnostic criteria and reference tables used by different countries. There have been attempts to get consensus and standardisation for testing.³⁰⁶ It would be vital to reach a national consensus before a screening programme could gain widespread acceptance.

The treatment³⁰³

According to NSC

1. 'There should be an effective treatment or intervention for patients identified through early detection, with evidence of early treatment leading to better outcomes than late treatment.'
2. 'There should be agreed evidence based policies covering which individuals should be offered treatment and the appropriate treatment to be offered.'
3. 'Clinical management of the condition and patient outcomes should be optimised in all health care providers prior to participation in a screening programme.'

This is probably the most controversial of all the areas and the biggest barrier to the implementation of a screening programme for COPD. Some authorities maintain that the research base of treatments for clinically detected COPD are unlikely to be applicable to those detected by a screening programme.³⁰⁰ There

is general agreement that treatment has some value in those with COPD detected after presentation with symptoms.³⁰⁷ The logic is that most patients detected by prevalence studies have mild or moderate disease.⁵³ In the lung age study there were no newly diagnosed cases of severe COPD. A quarter of new diagnoses were in the moderate category. Therapeutic interventions with medication, oxygen and pulmonary rehabilitation are mostly effective in those with moderate and severe disease. Furthermore, there is no evidence that drug treatment of COPD slows down decline in lung function.³⁰⁸ These two factors have lead to the conclusion that early detection and intervention is not useful.³⁰⁹ It is also argued that early detection in the mild or asymptomatic phase could lead to the excessive and unnecessary use of monitoring, medication, and medicalisation of mild disease and a diversion of resources better used to treat those with more serious conditions.⁶¹

In my opinion these arguments are flawed. If the medical profession do not take seriously the early signs of COPD it is likely that the patient will also trivialise their situation. If they have already stopped smoking then there is nothing more to be gained in detecting mild airflow obstruction but for those who are smoking or frequently relapse this sends the wrong messages as we know that their disease will almost certainly progress with continue smoking. An important part of primary care is about primary and secondary prevention and the early detection of disease as well as monitoring chronic illness. We already have a professional duty to diagnose early and monitor various conditions without actively treating them except with lifestyle advice. For example we monitor those with raised thyroid stimulating hormone (TSH) levels, borderline

hypertension, chronic kidney disease or obese people with impaired fasting glucose. Increased contact (annual or less) promotes the opportunity to reinforce brief health messages, prevent deterioration (with weight loss, exercise, diet or smoking advice) and detect the changes, which indicate the need for more active medical intervention. In the case of early COPD, once detected by screening, these individuals can be offered smoking cessation advice repeatedly and may benefit from other measures like influenza immunisation. Repeated testing will also allow earlier recognition of progression to a more serious level of disease requiring medication.

With good clinical guidelines and training it is unlikely that medication will be introduced prematurely and unnecessarily especially as it is well established that the course of the disease is not influenced by medication. At present medication is mainly used for relief of symptoms or long-term reduction in exacerbations. The first will be a relief to some patients who think they are just getting older or are unfit and the latter is only relevant under well-established criteria of disease severity and occurrence of recurrent infections. Therefore in my view it is unlikely that medication would be overused with earlier diagnosis.

The concerns over wasting resources may assume that highly paid doctors are doing the testing. There is plenty of evidence to suggest that other members of the primary health care team can be trained to perform spirometry even if interpretation has to be done by others.⁵ Moreover it is already assumed in the NHS that primary care personnel are using spirometry to diagnose and monitor those with COPD. Even in the absence of the new evidence described in this

thesis it is well known that simple brief advice from a health professional increases the chance of smoking cessation. Those with early lung damage are revealed as the group at risk of accelerated deterioration of lung function compared to non-susceptible smokers or non-smokers.⁶ Admittedly, once sustained abstinence from smoking has been achieved there is less indication for repeated screening or monitoring. It is not known if those with mild or asymptomatic COPD would benefit from other preventative care like annual influenza vaccination.

The second argument is that early detection of COPD with spirometry alone or as part of a multi-component intervention has not been shown to improve smoking cessation rates.^{151;159} These arguments have some merit but in my opinion are short sighted. Newer studies are pointing towards greater smoking cessation rates in those with lung damage as discussed previously (see Section 3.4.3, p. 84).¹⁵⁷

The screening programme³⁰³

According to NSC

1. There should be evidence from high quality Randomised Controlled Trials that the screening programme is effective in reducing mortality or morbidity. The information that is provided about the test and its outcome must be of value and readily understood by the individual being screened.
2. There should be evidence that the complete screening programme (test, diagnostic procedures, treatment/ intervention) is clinically, socially and

ethically acceptable to health professionals and the public

3. The benefit from the screening programme should outweigh the physical and psychological harm (caused by the test, diagnostic procedures and treatment)
4. The cost of the screening programme (including testing, diagnosis and treatment, administration, training and quality assurance) should be economically balanced in relation to expenditure on medical care as a whole (ie. value for money).

There may be some barriers to introduction of a screening programme from the public. The programme has to be acceptable to the public if uptake is to be sufficient to warrant the administrative effort involved. Identifying the population to be screened would rely on having access to a database of smokers and ex-smokers. Initial screening could be done through questionnaire about previous smoking and early symptoms which would increase the yield from spirometry.⁵ Inevitably, the screening will produce false positives. The psychological effects of this are unknown and need further research as outlined in Section 8.3.2 (p. 215).³⁰⁰

Economic analysis has been done to some extent with evidence presented in the NICE guidelines for COPD.³⁷ The model used showed that opportunistic case finding in primary care is a relatively cost-effective strategy but this has not been extended to screening per se.

If the anticipated benefits for those with COPD are viewed only in terms of long

term outcomes from drug therapy then I agree that screening is an inefficient method of detection and a waste of limited resources because the numbers needed to screen are estimated to be about 800 if smokers over 40 years of age are screened to defer the first exacerbation, which is a fairly innocuous positive outcome.³⁰⁰ However, if the results of this thesis are accepted and duplicated then the improvement in smoking cessation could be a major step forward in helping to improve long term morbidity from smoking.

In conclusion, I echo the British Lung Foundation's disappointment that screening was not introduced in 2008. They call the undiagnosed COPD patients 'the missing millions' which could have been detected if the British Government had included COPD when it announced new screening programmes in 2008.³¹⁰ However there is still the possibility that the new National Service Framework for COPD due late in 2008 will include changes in favour of earlier detection of COPD (see Section 8.7.3, p. 235).

8.7.3 National Service Framework

The background to National Service Frameworks (NSF) for COPD has already been outlined in Section 2.3.2 (p. 30). I submitted evidence from this research to the NSF External Reference Group (NSF-ERG) to support my recommendations to development of the NSF for COPD (Appendix 6 p. 311).

The NSF is due to be published and is likely to include a two-stage approach to screening for the disease. The first step would be a screening questionnaire and the second step would be spirometry for high-risk individuals. In my opinion it is imperative that the screening questionnaire does not rely purely on

subjective assessment of symptoms as many people attribute their reduction in exercise tolerance to poor general fitness or age and some will deny symptoms, which are observably present. There is strong evidence that questionnaire screening for smoking history and cough are strong predictors of abnormality on spirometry.⁵ The NICE guidelines for COPD already support opportunistic testing of those smokers and ex smoker who have symptoms and I hope the NSF takes this a step further towards a more organised strategy.³² The main pros and cons of screening for COPD have been discussed in the previous section (see p. 227).

9 Reflective section

9.1 Relationships with practices

The initial approach to practices was on the basis of e-mail and letter contact to a named doctor to ask whether their practice computer data included a list of current smokers and if they could easily give an indication of numbers of smokers recorded within the preceding twelve months over the age of 35 years. The response to this informal enquiry was encouraging. The pilot study had also given information about recruitment rates and strategy (see Section 9.2, p. 239).

I liaised with the director of public health at the Primary Care Trust to see what interest could be generated and what assistance could be available. The response was that they could not help with funding but they had an interest in improving knowledge, skills and training for primary care spirometry. Therefore they agreed to fund the purchase of ten spirometers of the type used in the research. They invited local practices to apply for the PCT spirometers with several provisos. The main condition was that a member of the practice would receive compulsory training in the theory and use of the equipment. Priority would be given to those practices who agreed to help recruit participants for the research project but involvement was not deemed mandatory.

The second wave of training and recruitment of practices to help in finding participants occurred through a primary care training day. At that time the Primary Care Trust was holding occasional half-day training sessions in topics

relevant to all primary care staff members. Each person elected to rotate through an optional series of 45-minute sessions about diagnosis and management of COPD including an opportunity to learn about the advantages of involvement with the research project. Dr Richard Dent, local respiratory consultant and clinical supervisor for this project, gave the preliminary plenary introductory talk and held a further optional question and answer session on COPD management at that training day. I gave a talk about the new general practice contract and COPD and how the participation in the research project could benefit diagnosis and data collection for the practice and their patients. Another one of the sessions on the rotation on that training day was practical hands-on spirometry by an experienced respiratory nurse.

Several practices were keen to participate and some were adamant that they wanted no involvement. Selected members of the research management team and I made formal visits to several practices which had shown some interest in the project, but were either undecided or wanted more detailed information. On three occasions I gave a detailed presentation to a group of practice personnel, including management, doctors, nurses and health assistants.

Each of the practices signed a contract with us to say that they had read and understood the information and would comply with the terms of the ethics committee. As with individual participants, it was made clear that a practice could drop out at any time should they change their mind. No practice dropped out and we did not have any problems or disputes with the practices involved. In fact relationships have been enhanced and communication is very good.

I produced an information pack for each of the five practices with details including ethics, procedures and resources such as templates for letters to send out to potential participants. Further training in searching computer databases and sending out invitation letters was given in-house at each of the five participating practices. Support and advice was available throughout and relationships remained good throughout the study period.

9.2 *Problems with recruitment*

Issues over recruitment are inevitable. The pilot study gave us a clear indication of how we could achieve the numbers necessary and the preliminary work on practice smoking registers indicated the number of practices, which would need to be involved to reach the targets.

Personal letters to individual smokers followed by a telephone call from the practice staff, supported by clinicians on an opportunistic basis, improved the numbers of willing participants from 4% to 18%.

I had very little adverse feedback from this process. One person phoned the research desk number to complain about privacy. All the letters were sent to smokers in the name of their own registered doctor on headed practice paper of that particular practice. The address for their response was the central research practice address. Once the person realised that we had no access to their personal records and the letter had been sent out from their own doctor they were satisfied that there had been no breach of confidentiality.

The funding body (The Health Foundation) for the project had given us a time limit for the award of 24 months from start to finish. This concentrated the efforts for recruitment but also created a bit of a problem as we reached the twelve-month mid-point without managing to recruit the 600 participants originally planned. I renegotiated the time frame with the Health Foundation and they were happy for a three-month extension on the basis that I was not asking for extra funds and that the statistical power of the study relied on sufficient participants. With the extension we were able to reach a modified target above 560 participants. As the original power calculation indicated the need for a total of approximately 300 participants with an overly pessimistic estimate for the attrition rate (see Section 6.1.2, p. 182), the actual recruitment level of 560 current smokers represented a generous margin of safety to achieve adequate follow up for statistical analysis.

9.3 Changes to protocol and ethical permission

The pilot study had exposed a number of issues that required modification for the full research study.

9.3.1 Abnormal results

It was important that the protocol made provision for results that necessitated review by the person's own doctor where the results of spirometry indicated an important abnormality other than smoking related airways obstruction. In consultation with R D (RD= consultant respiratory physician and clinical supervisor), I introduced new safety measures after the initial pilot study

highlighted the need. The safety net for these included a number of steps as follows. I reviewed all results of spirometry personally within two weeks of the initial data collection. If the spirometry was indicative of a restrictive disorder I sent the tracing and background clinical data to RD. If he confirmed my diagnosis I sent a letter directly to the participant asking them to make an appointment with their doctor. I also sent a letter to their doctor with a copy of the spirometry and a standard guideline of what conditions in their past medical history might be responsible (see Appendix 5, p. 308). The general practitioner was advised that their patient would be making an appointment and requested that the doctor should review the records to decide if this abnormality was already on record and also advised on what investigations or referral was necessary. We had no complaints from either the study participants or from their doctors about the way that this was conducted.

The original protocol was based on the British Thoracic Society guidelines for diagnosis of COPD. Under those guidelines it is necessary for the FEV1 to be under 80% of the predicted level (for age height and gender) and for the ratio of FEV1/FVC to be 70% or less. At that time the new General Medical Services contract also included the need to perform reversibility testing on those suspected of having COPD. Several issues arose to make this unsatisfactory. The National Institute for Health and Clinical Excellence (NICE) produced a new set of guidelines in February 2004 dismissing the requirement for reversibility testing. In many ways this change to national guidelines was irrelevant to this study. The main objective of the study was to see the effects of communicating lung damage through using the lung age concept and therefore the pros and

cons of using reversibility had limited relevance. However, part of the overt benefits of this study to participants and their doctors was to enhance and improve the records and registers of those with smoking related damage. Therefore our data had to be consistent with the new contract requirements (see QOF, p. 31) and indirectly and long term with NICE.

While inconsistencies existed between NICE and QOF we had to make a compromise in keeping with the aims of the study. In essence we wanted to get a 'personal best' result for individuals from spirometry. Dr RD suggested that to get a personal best for all participants regardless of initial raw testing we should give 400mcg of salbutamol to every participant. This could have also had the added benefit of streamlining the protocol. I had two objections to this idea.

The first objection was that the average time for the data collection and testing would be extended by about 20 minutes as this is the lag time needed from administration of salbutamol until its effect. Under the original protocol only one in four participants needed this extended time for testing.

The second problem with this approach is that we would have been giving an unnecessary drug to people who had normal lung function (in 75% of participants). This may have been more difficult to get through the ethics committee as an amendment and may have made smokers less willing to participate.

Therefore, because of the ethics of giving unnecessary medication and the

potential detriment to recruitment I took a pragmatic decision to make a minor change to the protocol. Any smoker who had an FEV1 under 80% (regardless of FVC result) would be given the dose of salbutamol recommended by BTS and retested after a 20 minute interval. A review of the cases where this would have been effective from the beginning was done to see if this had made any difference to the information given to participants. The conclusion was that the differences were subtle and of no relevance to the main aims of the study and primary outcome measure.

The protocol for spirometry, reversibility testing and referral lead to some interesting situations and required discussion with RD.

Example 1.

A 67-year-old man had been smoking for 54 years (since the age of 13). First spirometry indicated mild obstruction. His last cigarette had been 30 minutes before testing. After 400mcg of salbutamol his FEV1 had improved 10% and was subsequently in the normal range.

Commentary on example 1.

Under BTS guidance and by the modified quality framework (2008/09 new general medical services contract) for primary care this is a normal result. It demonstrates the value of the 'personal best' policy of the research and the usefulness of the European Respiratory Society guidance for COPD that uses 'post-bronchodilator' spirometry (see Section 3.1.1, p 39). Ironically it also illustrates the fact that despite 56 years of smoking and an estimated 68 pack years his lung function was normal. However he did have hypertension and

heart disease and had not thus far been persuaded to stop smoking.

Example 2.

A 77 year old man admitted to having diabetes, asthma and angina during the data entry interview and denied having any of the exclusion criteria. His initial spirometry performed on 31.1.2005 suggested a restrictive disorder. His FEV1 was 31% of the predicted value with a normal FEV1/FVC ratio. He was in the control group and therefore lung age was not given but as he had a restrictive pattern on spirometry his GP was alerted in keeping with the protocol.

Commentary on example 2.

Restrictive disorders or impairments are characterised by reduced lung expansion and lung volumes compared to predicted normal values. Typically because both the forced expiratory volume (in the first second) and vital capacity are reduced the ratio of the two values is normal on spirometry. Any condition reducing diaphragm movement may cause a restrictive pattern including pregnancy, tight corsets or central obesity. Poor function of the other respiratory muscles due to injury or disease can also cause restriction. Disease of the lung tissue such as asbestosis, pulmonary fibrosis, sarcoid, or pleural effusion can also cause a restrictive pattern.

Asbestosis was one of the exclusion criteria in the recruitment protocol but had not been recorded so far in this man's history. He said he had asthma but review of his notes for this thesis (he is a registered patient at my practice) shows that his GP had thought he had COPD. The patient was subsequently investigated by the local physicians and in 10.7.2007 (2 years after recruitment

to the research) finally had a high resolution Computerised Tomography scan which showed he had interstitial lung disease and a history and findings consistent with asbestos damage. The research had correctly picked up an abnormality that previous contact with clinicians had not interpreted correctly and the safety net of referral mechanisms had worked successfully. Despite having multiple medical problems made worse by smoking he is still alive and continues to smoke at the time of writing (October 2008).

9.3.2 Outcome measures

Change in lung function

A change in FEV1 from recruitment compared to follow up was a secondary outcome in the original pilot study. This was used as an incentive to draw participants back who were not in the intervention group. The control group were told:

‘Whether or not you have stopped smoking we will invite you back after 12 months to see if there are any changes to your lung function’. (see Appendix 5. Information sheet for participants, p. 303)

This information was then included in their personal letter after the follow-up examination. In reality this short-term data was not regarded as an important outcome as previous research had established that FEV1 measurement in COPD does not change significantly year on year but on the surface it seemed an acceptable reason for encouraging those who were in the control group to return for follow up.^{61,63} However, the collection of this data resulted in a few interesting clinical situations. Below, I will outline an example which demonstrates the real life general practice beneath the statistics.

Example

Mr A was aged 49 at recruitment and was smoking 15 cigarettes per day with a history of 23 pack years. He had no exclusion criteria and was found on spirometry to have a lung age of 113. The FEV1 was 1.65 litres which was only 43 % of the predicted level for a man of his age and height. There was no improvement 20 minutes after giving four puffs of salbutamol metered dose inhaler through a spacer device. Later, further interpretation of his results showed him to have a restrictive pattern. He was referred to his GP for further evaluation where he was noted to be morbidly obese (weight 165kg and BMI 51) he also had an abnormal glucose tolerance test consistent with diabetes mellitus. On follow up after twelve months he was still smoking but his lung age had improved dramatically and was now 64 years (FEV1 3.63 litres 76% of predicted). Therefore his forced expiratory volume in the first second had improved by nearly two litres.

Commentary on example

There is a danger that the narrative of illness is lost in the facts and figures of quantitative research. This example illustrates one of the detrimental effects of gross obesity. On review of his records after one year from recruitment his lung function was very much improved and his weight had dropped to 128 kg (BMI 39). Although he remains grossly obese, he said he attributed his improved lung function to the loss of 40kg of weight and felt much better. It is not possible to attribute the loss of weight to the intervention in this research but it is encouraging that this has been an unintended outcome. Moreover, his last set of blood test results indicates that his fasting blood sugar and glycosylated haemoglobin (HbA1c) have returned to normal.

This was an exceptional case but demonstrates that data collection and clinical context is important when considering the outliers in terms of odd results. In this case an unprecedented improvement in FEV1 needed an explanation and it was fortunate that we had access to the information. The pros and cons of recording weight and collecting other data and measuring other outcomes not included in this study will be discussed in the section on weakness of the study (see p. 207).

Measuring cotinine

The original protocol for the pilot study included the measurement of exhaled carbon monoxide as the only objective method of confirming self-reported smoking cessation. It did not include an independent collection and analysis of objective biochemical confirmation of smoking cessation with cotinine levels. The initial proposal submitted to the funding agency also did not contain cotinine measurement. As part of the conditions for funding the Health Foundation insisted that independent confirmation should be included and funding was supplied. Ethics approval was sought and easily obtained as a modification of the original protocol. Self-reporting of abstinence from smoking is not necessarily reliable and the integrity of research results is greater when independent biochemical validation is conducted and is discussed in detail elsewhere in this thesis (see p. 145).^{141;153}

Drug interactions

Interactions of salbutamol with other medication are extremely rare but there

were several situations where there was a theoretical interaction of beta blockers with the beta agonist (salbutamol). RD maintained that the interaction would not be of clinical significance and we would not be compromising patient safety with simultaneous use. Also, in theory beta-blockers may make airways narrower (by bronchospasm in asthmatics) and therefore make lung age or FEV1 worse. The protocol would have detected and briefly countered those effects without needing any changes.

9.4 *What is the meaning of lung age?*

During a conference presentation of the research at a fairly early stage at a primary care research conference on 3rd March 2005, I was challenged by a member of the audience about the meaning of lung age. It is interesting that during the hundreds of times I had explained the lung age graph nobody else had made this challenge. The point was – ‘What does it mean to have the lungs of a 75 year old? Most fit 75 year old do not have breathing trouble and are not compromised or concerned about their lungs so why should we be?’

Estimates of lung age made by professionals have not been used before in research and personal estimates made by smokers about their own health are a new concept with research in its infancy.⁵⁶ No published qualitative research is available about the perceived meaning and impact of this information. Since the completion of the study one pilot study has been done on the psychological factors which included a preliminary personal estimate of lung age by the participants.⁵⁶ Suffice it to say that further research is needed and this has been explored in Section 8.3.1 (p. 211).

Some concern was raised when we discovered that some participants were related to each other. What would we do if they were randomly allocated to different groups? Would there be problems with contamination? In reality this did not happen often. There are a couple of examples of married couples who were allocated to different groups. Therefore one knew their lung age and the other did not. On another occasion both were allocated to the same group and a healthy competition arose.

9.5 Reflections on BMJ rapid responses

The original published British Medical Journal article is reproduced in Appendix 7 (p. 312), the accompanying BMJ editorial in Appendix 8 (p. 319) and the full text of the rapid responses is reproduced in Appendix 9 (p. 320).

Professor Hiroshi Kawane from Japan expressed concerns that lung ages above 100 years made using lung age 'impractical' (see Rapid response on 7th March 2008, p. 320). I think that he is suggesting that the patient cannot extract meaning or understand from this type of information. The assumption that he makes is that there is no point in giving results like this to smokers. However, I think that is a question that has not been answered by any published research and leads to some interesting questions that should be the subject of proposals for future research (see Section 8.3, p. 210).

A second letter (see Rapid response on 8th March 2008, p. 320) seemed to criticise the absence of a counselling arm of the research. It is completely true that the study did not attempt to compare more complex counselling. Those

who volunteer for repeated and time-consuming counselling are probably, by selection and self-selection, a different group to those who were willing to volunteer for this study and by definition in a group who want to quit. This research is indeed attempting to see the effect of a short 'one-off' intervention on twelve-month outcomes. This highlights the need for the inclusion of other data about the research population in different studies so that meaningful comparisons can be made.

A lay correspondent requested a chart or scale to be made available with the idea that individual members of the public can do their own check in the same way as peak flow meters are used by asthmatics to monitor their own peak expiratory flow rate (PEFR) (Rapid response on 9th March 2008, p. 320). This opens up an interesting area of potential discussion about how results are interpreted (in this case by a non medical person) and used. There are at least two areas of misconception encapsulated in this response. The first misconception is that peak flow rates and other spirometry results are related. My answer was that some attempts have been made by other researchers to correlate FEV1 with PEFR but this has never become an established method. Therefore there is no reliable formula to make the conversion from PEFR into lung age. There are well-established diagnostic criteria for diagnosing COPD using spirometry measurements (FEV1 and FEV1/FVC ratio) but PEFR cannot be used for this purpose. PEFR is very valuable for diagnosis and monitoring of asthma.³² I believe that I missed an opportunity to address a broader question, which has at its centre a second misconception. The writer of the letter is assuming that the research results can be extrapolated to mean that there value

in smokers self-testing their lung age. Although there has been a move towards asthmatic patients taking greater control of their condition by regular self-testing of PEFr there is no published research to support self-testing of FEV1 in smokers or COPD. Chronic disease management is well ingrained in the primary care system in the NHS but there is a paucity of evidence to show the benefit of regular monitoring of lung function.^{311 61}

Drs M and S Saraswat made some good observations about the difficulties of generalising the results to other populations (Rapid response on 9th March 2008, p.320). In particular they were concerned about the selected population and lack of inclusion of those with mental health problems. Certainly these were two of the weaknesses of the study. The protocol did not exclude those with mental disorders or other addictions. However the data collection did not collect information specific to mental health or socio-economic status. Anecdotally we were very aware that we recruited some people with depression. This will be discussed at greater length under a later section about weaknesses of the study (see p. 205).

The long response from Jay Schrand (27th March 2008, p. 320) discusses among other things the possible effect of damage to the part of the brain called the insular and likelihood of smoking cessation. Among the number of points that he tries to make is that because the control group had a significantly larger number of stroke patients and because stroke in the insular region of the brain promotes disgust for previously enjoyable stimuli the effect of the intervention in the lung age study may have been diluted. It is indeed true that the intervention

group had a larger number of participants with cerebro-vascular accidents (CVA) than the control group. In a sub-analysis I have looked at whether those with a history of CVA are more likely to respond to the intervention. The results section does not portray the full clinical picture of what happened during the trial. The baseline characteristics are that 11 (4.2%) of the control group gave a self-reported past history of stroke or transient Ischaemic attack whereas the intervention group had a significantly lower number (2 or 0.8%) (see Table 5, p. 194). These numbers changed at follow up. During the twelve-month follow up period, the control group acquired one more person to the CVA/TIA group and the intervention group more than doubled to five people with CVA/TIA. This in itself is an impressive demonstration of the risk of complications from smoking. Only one person from each group was among the successful quitters.

There are several flaws to Mr Schrand's arguments. First and foremost if stroke was instrumental in reducing smoking then this would have happened in the immediate aftermath of the event, not at some distant future date of another intervention. Therefore those people would have already stopped smoking and not even appear in this study of continuing smokers. Secondly the study was not designed to analyse the role of stroke in smoking cessation and therefore the changes observed within this subgroup cannot be regarded as significant even though the fact that one person from each group stopped smoking is interesting from an illness narrative point of view. In fact hidden within the quantitative data is the interesting observation that most of the people in this group had no intention of quitting or trying to quit at the time of recruitment to the study. This is no surprise as they are a group who must be very resistant to

health messages from professionals especially as some had other co-morbidities such as coronary heart disease and diabetes. The 77 year old in the control group who quit was already at the action stage of change. The person in the intervention group also was in the action stage of change when recruited and had a poor lung age (93 years) for her actual age of 69. Unfortunately she was one of those who had a stroke during the study period. The datum does not reveal which of these factors stimulated her ultimate smoking cessation. This is an area that needs further research (see Section 8.3, p. 210).

A public health trainee made some very useful observations about the precise nature of the intervention highlighting the fact that lung age is not given in isolation to an interaction with a professional and more contact than the control participants which may have led to bias (see Rapid response on 28th March 2008, p. 320). My response was to agree with their statement that the intervention is more than just giving lung age measurements. However, it is not correct to say that this is methodological bias. Bias is defined as 'any systematic error in the design, conduct or analysis of a study that results in a mistaken estimate of an exposure's effect'.³¹² Although the abstract summarized the intervention as 'giving results in terms of lung age', they erroneously failed to look beyond the abstract to fully understand the methodology of the research. The full text version of the research intervention clearly and openly states that: 'Participants in the intervention group were given their results verbally, immediately after randomisation, in the form of 'lung age' with a graphic display'. And 'Written results were given to the intervention group as lung age'. This is not bias, this is the complex intervention. Anyone wanting to introduce

the intervention in their own clinical practice must be careful not to shortcut the intervention to just a verbal delivery of 'lung age'. The complex intervention that produced the effect on quit rate in this study has three important components: verbal information, graphic information, and an individualised results letter. Together, these interventions seem to help people think about their health in a personalised way. It is not possible to isolate the component of this study that had the greatest influence. The misunderstanding of this respondent emphasises the point that clinicians, managers and commercial interest groups will need to consider the 'whole package' if they wish to get similar favourable outcomes.

Dr Donzeli, Director of Health Education Service in Milan, objects to my recommendation that lung age testing in smokers could be included in the new General Practice Contract for the England (Rapid response on 8th April 2008, p. 320). I agree that incentives in the form of targets and financial inducements for activity can be a poor substitute for payment for results. From an economic perspective the controllers of the purse strings would probably be better off only paying clinicians for proof of sustained cessation. However, the current system in NHS smoking cessation clinics of declaring success rates at four weeks post treatment are at best naïve and at worst a cynical way of over-inflating the achievements of the strategy. As Dr Donzeli works in a different health system his perspective is influenced by his own working environment. My suggested strategy to improve diagnosis of COPD and, as a potential spin-off, improve cessation rates was made in the context of an existing computerised data collection system and payment structure.

Philip H. Quanjer, who was the main author of some authoritative papers from the European Respiratory Society on standardisation of lung function testing,³⁰⁶ asks the question 'Should we use lung age?' (see Rapid response on 16th April 2008, p. 320). He uses the example of a 50 year old 1.78 metre tall man. His first point is that different reference tables (ECCS/ERS³⁰⁶, Crapo and Morris⁶⁴)³⁰⁶ for assessment of spirometry results each have a different average result for the predicted FEV1 of an individual, which vary by a factor of over ten percent (350mls). He makes the further observation that if this subject had the exact average FEV1 under each of these three different reference tables his lung age would be between 62 and 83 years. He therefore regards the concept of lung age as fictitious because it does not take into account the concept of a range of normal. He goes on to question the desirability of using this 'fiction' within the context of the doctor-patient relationship.

I have to agree with Dr Quanjer that we have an interesting dilemma. It is true that the different tables of normal and abnormal test results given by different authorities lead to a wide range of normal. It is also true that my research is using the concept of lung age to provoke change. It is open to debate whether this constitutes fiction, deception or legitimate use of the concept. Even if a single set of reference tables is analysed in more depth it is apparent that there are some interesting observations. The BTS and NICE both regard an FEV1 reading of more than 80% of predicted result as normal, using the ECCS tables as its reference (see Table 3, p. 41). Given that lung age is calculated as equivalent to 100% of predicted value, values for our subject of between 80%

and 99% will give a lung age that is different (worse) compared to the chronological age. This is an inevitable consequence of using this concept, which may be regarded as a useful strategy or deception depending on perspective. This is also discussed under the ethics section (see p. 257).

The other issue highlighted here is the international differences in criteria for diagnosis of abnormal and the different reference tables. With reference to the example of a 50-year-old 1.83 metre tall male, according to ECCS his results will be regarded as 100% normal if his FEV1 is 3.86 litres. The same subject tested by ATS guidelines using the NHANE 3 reference tables would have 100% normal results if his FEV1 is 3.29 litres.⁶⁵ There has been a call for standardisation between countries (with the caution that different racial groups need adjustments to the calculations) and concerns about the variable definitions of abnormal spirometry.^{306;313} The potential confusion does not end there as other authors question the accuracy of using a percentage of predicted level or an absolute level for the ratio of FEV1/FVC to make a diagnosis of COPD. They regard a better measure using standard deviations from predicted normal. Further discussion of the complexities of different reference tables and accepted normal measurements in different countries is beyond the scope of this thesis.

9.6 *Ethical issues*

From the start of the protocol development I was conscious of the possible problems of giving bad news (about lung function) that might offend or upset and of giving good news, which might encourage the continuation of harmful behaviour. The ethics committee did not bring up any objections at all about this aspect of the research. The feedback to participants was very carefully worded in an attempt to avoid harm. The immediate feedback and the personalised letters to individuals sent after testing made it clear that even in the context of 'normal results' we still encourage smokers to give up smoking. The results do not give any information about the other effects of smoking such as the risk heart disease, lung cancer and stroke. In other words normal lung testing did not mean everything else was fine.

The mental health of individuals was not measured. I did become aware that a number of smokers attending the research were also on antidepressants. Depression and the use of antidepressants were not on the list of exclusions for the study. Subsequent further literature review of previous research reveals a mixture of literature with some associations of smoking, depression and relapse²⁹¹ and some which show improvements in mental health with quitting.³¹⁴ Therefore, in hindsight it may have been wiser to exclude those with moderate or severe depression in case the news of lung age was detrimental to the state of their mental health. However, this was not done and no negative feedback has been received through the local general practitioners.

Many studies, of interventions designed to promote smoking cessation, include measures of depression and some are specifically targeted at those with depression. I acknowledge that some useful data could have been generated had this study included a depression score in the baseline and follow up data collection. This is discussed more under the section on weakness of the study (see Section 8.2.2, p. 207).

The ethics of giving good news to those with normal lung function is possibly the reverse side of the same coin. In essence this is the negative side of the question 'does bad lung age encourage people to stop smoking?'

Research evidence published since the start of this project seemed to indicate that poorer lung function is correlated with increased quit rates.¹⁵⁷ In the absence of that evidence and without any data at all about the effects of giving data as lung age any outcome was possible and the only means of testing the theory was with a randomised controlled trial. The default message to participants was to encourage them all to quit regardless of the results and with the message that the lung function was not the only effect either way. Therefore I believe ethically our position was sound.

10 References

Reference List

- (1) WHO. Management of substance abuse. Internet [2008 Available from: URL:http://www.who.int/substance_abuse/facts/tobacco/en/index.html
- (2) WHO Europe. The European Tobacco Control Report 2007. World Health Organization Regional Office for Europe, editor. 2007. Publications WHO Regional Office for Europe Scherfigsvej 8 DK-2100 Copenhagen Ø, Denmark. 23-6-2008. Ref Type: Report
- (3) Callum C. *The UK smoking epidemic: deaths in 1995*. 1998. London: Health Education Authority. Ref Type: Report
- (4) Anderson JE, Jorenby DE, Scott WJ, Fiore MC. Treating tobacco use and dependence: an evidence-based clinical practice guideline for tobacco cessation.[comment]. Chest 2002; 121(3):932-941.
- (5) Van Schayck CP, Loozen JM, Wagena E, Akkermans RP, Wesseling GJ. Detecting patients at a high risk of developing chronic obstructive pulmonary disease in general practice: cross sectional case finding study. BMJ 2002; 324:1370.
- (6) Fletcher C, Peto R. The natural history of chronic airflow obstruction. British Medical Journal 1977; 1(6077):1645-1648.
- (7) Kuller LH, Ockene JK, Meilahn E, Wentworth DN, Svendsen KH, Neaton JD. Cigarette smoking and mortality. MRFIT Research Group. Preventive Medicine 1991; 20(5):638-654.
- (8) Hackshaw AK, Law MR, Wald NJ, Hackshaw AK, Law MR, Wald NJ. The accumulated evidence on lung cancer and environmental tobacco smoke.[see comment]. BMJ 1997; 315(7114):980-988.
- (9) Law MR, Morris JK, Wald NJ. Environmental tobacco smoke exposure and ischaemic heart disease: an evaluation of the evidence. BMJ 1997; 315(7114):973-980.
- (10) Pitsavos C, Panagiotakos DB, Chrysoshoou C, Skoumas J, Tzioumis K, Stefanadis C et al. Association between exposure to environmental tobacco smoke and the development of acute coronary syndromes: the CARDIO2000 case-control study. Tob Control 2002; 11(3):220-225.

- (11) Panagiotakos DB, Pitsavos C. Passive smoking's role in diabetes. *BMJ* 2006; 332(7549):1044-1045.
- (12) Cook DG, Strachan DP. Health effects of passive smoking. 3. Parental smoking and prevalence of respiratory symptoms and asthma in school age children. *Thorax* 1997; 52(12):1081-1094.
- (13) Lumley J, Oliver SS, Chamberlain C, Oakley L. Interventions for promoting smoking cessation during pregnancy. Lumley J, Oliver SS, Chamberlain C, Oakley L Interventions for promoting smoking cessation during pregnancy The Cochrane Database of Systematic Reviews: Reviews 2004 Issue 4 John Wiley & Sons, Ltd Chichester, UK DOI : 10.1002 /14651858 CD001055 pub2 2004.
- (14) Department of Health. Smoking Kills. A White Paper on Tobacco. 1-1-1998. TSO.
Ref Type: Report
- (15) Office for National Statistics. *Population Trends Number 93, Autumn 1998*. London. 1998. The Stationery Office.
Ref Type: Report
- (16) Peto R LABJea. Mortality from smoking in developing countries 1950-2000. Oxford. 2007. Oxford University Press, 1994.
Ref Type: Report
- (17) Buck D GCPSRM. *Cost effectiveness of smoking cessation interventions*. 1997. University of York Centre for Health Economics., Health Education Authority.
Ref Type: Report
- (18) NHS The information Centre. Statistics on smoking: England,2008. Internet [2008 [cited 2008 Nov. 11]; Available from:
URL:<http://www.ic.nhs.uk/statistics-and-data-collections/health-and-lifestyles/smoking>
- (19) Drever F WMe. *Health inequalities: decennial supplement: Office for National Statistics*. (Series DS; no. 15). 1997. London: The Stationery Office.
Ref Type: Report
- (20) Siahpush M, McNeill A, Borland R, Fong GT. Socioeconomic variations in nicotine dependence, self-efficacy, and intention to quit across four countries: findings from the International Tobacco Control (ITC) Four Country Survey. *Tob Control* 2006; 15(suppl_3):iii71-iii75.
- (21) Fichtenberg CM, Glantz SA. Effect of smoke-free workplaces on smoking behaviour: systematic review. *BMJ* 2002; 325(7357):188.

- (22) Serra C, Cabezas C, Bonfill X, Pladevall-Vila M, Serra C, Cabezas C et al. Interventions for preventing tobacco smoking in public places. [Review] [56 refs]. Cochrane Database of Systematic Reviews 2000;(3):CD001294.
- (23) Lovato C, Linn G, Stead LF, Best A, Lovato C, Linn G et al. Impact of tobacco advertising and promotion on increasing adolescent smoking behaviours. [Review] [75 refs]. Cochrane Database of Systematic Reviews 2003;(4):CD003439.
- (24) ITVNews. Smoking ban 'helps people quit'. Internet [2008 [cited 2008 July 1]; Available from: URL:<http://www.itv.com/News/Articles/Smoking-ban-helps-people-quit-714345318.html>
- (25) Cicutti Nic. Is smoking a necessary evil? on line [2008 Available from: URL:<http://money.uk.msn.com/tax/articles/article.aspx?cp-documentid=4750489>
- (26) Butler C C, Rollnick S, Cohen D, Bachman M, Russell I, Scott N. Motivational consulting versus brief advice for smokers in genreal practice: a randomized trial. Br J Gen Pract 1999; 49:611-616.
- (27) Hyland A, Li Q, Bauer JE, Giovino GA, Steger C, Cummings KM et al. Predictors of cessation in a cohort of current and former smokers followed over 13 years. Nicotine & Tobacco Research 2004; 6 Suppl 3:S363-S369.
- (28) NICE. Managing stable COPD. Thorax 2004; 59(90001):i39-130.
- (29) NICE. Brief interventions and referral for smoking cessation in primary care and other settings. NICE [2006 Available from: URL:<http://www.nice.org.uk/PHI001>
- (30) NICE. Hierarchy of evidence and grading of recommendations. Thorax 2004; 59(90001):i13-i14.
- (31) Scottish Intercollegiate Guidelines Network. SIGN Hierarchy of evidence. Scottish Intercollegiate Guidelines Network, Elliott House, 8-10 Hillside Crescent,Edinburgh EH7 5EA [2008 [cited 8 A.D. Sept. 20]; Available from: URL:<http://www.sign.ac.uk/guidelines/fulltext/50/annexb.html>
- (32) NICE. Diagnosing COPD. Thorax 2004; 59(90001):i27-i38.
- (33) Freeman D. COPD prevalence in asymptomatic smokers - The first 100. Prim Care Resp J 2000; 9:41.
- (34) Seamark D, Williams S, Powell R, Timon S, Ward A, Ward D et al. Home or surgery based screening for COPD. Prim Care Resp J 2001; 10(2):30-33.

- (35) Humerfelt S, Eide GE, Kvale G, Aaro LE, Gulsvik A. Effectiveness of postal smoking cessation advice: a randomized controlled trial in young men with reduced FEV1 and asbestos exposure. *Eur Respir J* 1998; 11(2):284-290.
- (36) Risser NL, Belcher DW. Adding spirometry, carbon monoxide, and pulmonary symptom results to smoking cessation counseling: a randomized trial. *Journal of General Internal Medicine* 1990; 5(1):16-22.
- (37) NICE. *The draft guidelines from NICE on COPD* . 2003.
Ref Type: Internet Communication
- (38) Crown. National service frameworks (NSFs). Department of Health [2007 Available from:
URL:http://www.dh.gov.uk/en/Policyandguidance/Healthandsocialcaretopics/DH_4070951
- (39) BMA. Quality and outcomes framework BMA. Internet [2006 [cited 2008 Sept. 1]; Available from: URL:www.bma.org.uk/ap.nsf/content/qof06-clinicalind~copd
- (40) Halbert RJ, Isonaka S, George D, Iqbal A. Interpreting COPD prevalence estimates: what is the true burden of disease? *Chest* 2003; 123(5):1684-1692.
- (41) Bandolier. COPD prevalence. Bandolier [2004 Available from:
URL:<http://www.jr2.ox.ac.uk/bandolier/band113/b113-3.html>
- (42) Murtagh E, Heaney L, Gingles J, Shepherd R, Kee F, Patterson C et al. Prevalence of obstructive lung disease in a general population sample: the NICECOPD study. *European Journal of Epidemiology* 2005; 20(5):443-453.
- (43) Anonymous. Managing stable chronic obstructive pulmonary disease.[erratum appears in *Drug Ther Bull* 2002 Jun;40(6):48]. [Review] [57 refs]. *Drug & Therapeutics Bulletin* 2001; 39(11):81-85.
- (44) Price D, Duerden M. Chronic obstructive pulmonary disease. *BMJ* 2003; 326:1046-1047.
- (45) Celli BR, Halbert RJ, Isonaka S, Schau B. Population impact of different definitions of airway obstruction. *Eur Respir J* 2003; 22(2):268-273.
- (46) Clotet J, Gómez A, X, Ciria C, Albalad JM. Spirometry is a good method for detecting and monitoring chronic obstructive pulmonary disease in high-risk smokers in primary health care. *Archivos de Bronconeumologia* , 2004;(155-159):-159.
- (47) British Thoracic Society. British thoracic society Clinical information. Internet [2004 [cited 2008 Nov. 5]; Available from: URL:<http://www.brit-thoracic.org.uk/ClinicalInformation/tabid/66/Default.aspx>

- (48) Geijer RM, Sachs AP, Hoes AW, Salome PL, Lammers JW, Verheij TJ. Prevalence of undetected persistent airflow obstruction in male smokers 40-65 years old. *Fam Pract* 2005; 22(5):485-489.
- (49) Doherty DE. Early detection and management of COPD. What you can do to reduce the impact of this disabling disease. [Review] [34 refs]. *Postgraduate Medicine* 2002; 111(6):41-44.
- (50) Calverley PM, Anderson JA, Celli B, Ferguson GT, Jenkins C, Jones PW et al. Salmeterol and fluticasone propionate and survival in chronic obstructive pulmonary disease.[see comment][reprint in *Ugeskr Laeger*. 2007 Sep 17;169(38):3198-201; PMID: 17910830]. *New England Journal of Medicine* 2007; 356(8):775-789.
- (51) Rabe KF, Rabe KF. Treating COPD--the TORCH trial, P values, and the Dodo.[see comment][comment][erratum appears in *N Engl J Med*. 2007 Apr 19;356(16):1692]. *New England Journal of Medicine* 2007; 356(8):851-854.
- (52) Nihlen U, Montnemery P, Lindholm LH, Lofdahl CG. Detection of chronic obstructive pulmonary disease (COPD) in primary health care: role of spirometry and respiratory symptoms. *Scand J Prim Health Care* 1999; 17(4):232-237.
- (53) Stratelis G, Jakobsson P, Molstad S, Zetterstrom O. Early detection of COPD in primary care: screening by invitation of smokers aged 40 to 55 years. *The British journal of general practice : the journal of the Royal College of General Practitioners*, , Mar 2004;-6.
- (54) Griffiths C, Feder G, Wedzicha J, Foster G, Livingstone A, Marlowe GS. Feasibility of spirometry and reversibility testing for the identification of patients with chronic obstructive pulmonary disease on asthma registers in general practice. *Respiratory Medicine* 1999; 93(12):903-908.
- (55) Miravittles M, Fernandez I, Guerrero T, Murio C. [Development and results of a screening program for COPD in primary care. The PADOCC Project(Program for the Increase in the Diagnosis of COPD in Primary Care).[comment]. [Spanish]. *Archivos de Bronconeumologia* 2000; 36(9):500-505.
- (56) Lipkus IM, Prokhorov AV. The effects of providing lung age and respiratory symptoms feedback on community college smokers' perceived smoking-related health risks, worries and desire to quit. *Addictive Behaviors* 2007; 32(3):516-532.
- (57) van der Meer RM, Wagena EJ, Ostelo RW, Jacobs JE, Van Schayck CP. Smoking cessation for chronic obstructive pulmonary disease. [Review] [52 refs]. *Cochrane Database of Systematic Reviews* 2003;(2):CD002999.

- (58) Ferguson GT, Enright PL, Buist AS, Higgins MW. Office Spirometry for Lung Health Assessment in Adults: A Consensus Statement From the National Lung Health Education Program. *Chest* 2000; 117(4):1146-1161.
- (59) Enright PL, Crapo RO. Controversies in the use of spirometry for early recognition and diagnosis of chronic obstructive pulmonary disease in cigarette smokers. [Review] [44 refs]. *Clinics in Chest Medicine* 2000; 21(4):645-652.
- (60) Enright PL, Kaminsky DA, Enright PL, Kaminsky DA. Strategies for screening for chronic obstructive pulmonary disease. [Review] [37 refs]. *Respir Care* 2003; 48(12):1194-1201.
- (61) van den Bemt L, Schermer T, van Weel C. Rational monitoring of COPD: where do current clinical guidelines stand? *Eur Respir J* 2007; 29(6):1078-1081.
- (62) ATS. American Thoracic Society. Internet [2008 [cited 2008 Oct. 28]; Available from: URL:<http://www.thoracic.org/sections/copd/index.html>
- (63) GOLD. GOLD. <http://www.goldcopd.org/Guidelineitem.asp?l1=2&l2=1&intId=1116> [2008 [cited 2008 Apr. 12]; Available from: URL:<http://www.goldcopd.org/Guidelineitem.asp?l1=2&l2=1&intId=1116>
- (64) Crapo RO, Morris AH, Gardner RM, Crapo RO, Morris AH, Gardner RM. Reference spirometric values using techniques and equipment that meet ATS recommendations. *American Review of Respiratory Disease* 1981; 123(6):659-664.
- (65) Hankinson J L., Odencrantz J R, Fedan K B. Spirometric Reference Values from a Sample of the General U.S. Population. *Am J Respir Crit Care Med* 1999; 159(1):179-187.
- (66) National Collaborating Centre for Chronic Conditions. NICE [2003 section 7.6.1.1.21:[p.75] Available from: URL:www.nice.org.uk/Docref.asp?d=84986
- (67) National Collaborating Centre for Chronic Conditions. Chronic obstructive pulmonary disease. National clinical guideline on management of chronic obstructive pulmonary disease in adults in primary and secondary care. *Thorax* 2004; 59 Suppl 1:1-232.
- (68) Calverley PM, Burge PS, Spencer S, Anderson JA, Jones PW. Bronchodilator reversibility testing in chronic obstructive pulmonary disease.[see comment]. *Thorax* 2003; 58(8):659-664.
- (69) Borg BM, Reid DW, Walters EH, Johns DP. Bronchodilator reversibility testing: laboratory practices in Australia and New Zealand.[see comment]. *Medical Journal of Australia* 2004; 180(12):610-613.

- (70) Department of Health. Proposed changes to the quality and outcomes framework for the financial year 2008/09. Department of Health [2008 [cited 2008 June 23]; Available from:
URL:http://www.dh.gov.uk/en/Publicationsandstatistics/Lettersandcirculars/Dearcolleagueletters/DH_083707
- (71) Vrijhoef HJ, Diederiks JP, Wesseling GJ, Van Schayck CP, Spreeuwenberg C. Undiagnosed patients and patients at risk for COPD in primary health care: early detection with the support of non-physicians. *Journal of Clinical Nursing* 2003; 12(3):366-373.
- (72) Morris JF, Temple W. Spirometric "lung age" estimation for motivating smoking cessation. *Preventive Medicine* 1985; 14:655-662.
- (73) Pride NB. Smoking cessation: effects on symptoms, spirometry and future trends in COPD. *Thorax* 2001; 56(90002):7ii-10.
- (74) He J, Vupputuri S, Allen K, Prerost MR, Hughes J, Whelton PK. Passive Smoking and the Risk of Coronary Heart Disease -- A Meta-Analysis of Epidemiologic Studies. *N Engl J Med* 1999; 340(12):920-926.
- (75) Critchley J, Capewell S. Smoking cessation for the secondary prevention of coronary heart disease. Critchley J, Capewell S Smoking cessation for the secondary prevention of coronary heart disease *The Cochrane Database of Systematic Reviews: Reviews* 2003 Issue 4 John Wiley & Sons , Ltd Chichester, UK DOI : 10 1002 /14651858 CD003041 pub2 2003.
- (76) Ford ES, Ajani UA, Croft JB, Critchley JA, Labarthe DR, Kottke TE et al. Explaining the Decrease in U.S. Deaths from Coronary Disease, 1980-2000. *N Engl J Med* 2007; 356(23):2388-2398.
- (77) Kenfield SA, Stampfer MJ, Rosner BA, Colditz GA. Smoking and Smoking Cessation in Relation to Mortality in Women. *JAMA: The Journal of the American Medical Association* 2008; 299(17):2037-2047.
- (78) Jarvis MJ. Why people smoke. *BMJ* 2004; 328(7434):277-279.
- (79) West R, West R. Assessment of dependence and motivation to stop smoking. [Review] [0 refs]. *BMJ* 2004; 328(7435):338-339.
- (80) Hyland A, Borland R, Li Q, Yong HH, McNeill A, Fong GT et al. Individual-level predictors of cessation behaviours among participants in the International Tobacco Control (ITC) Four Country Survey. *Tob Control* 2006; 15(suppl_3):iii83-iii94.
- (81) Curry SJ, Grothaus L, McBride C. Reasons for quitting: intrinsic and extrinsic motivation for smoking cessation in a population-based sample of smokers. *Addictive Behaviors* 1997; 22(6):727-739.

- (82) Duncan CL, Cummings SR, Hudes ES, Zahnd E, Coates TJ. Quitting smoking: reasons for quitting and predictors of cessation among medical patients. *Journal of General Internal Medicine* 1992; 7(4):398-404.
- (83) Frid D, Ockene IS, Ockene JK, Merriam P, Goldberg R, Kristeller J et al. Severity of angiographically proven coronary artery disease predicts smoking cessation. *American Journal of Preventive Medicine* 1991; 7(3):131-135.
- (84) Parkes G, Greenhalgh T, Griffin M, Dent R. Effect on smoking quit rate of telling patients their lung age: the Step2quit randomised controlled trial. *BMJ* 2008; 336(7644):598-600.
- (85) Crowley TJ, MacDonald MJ, Zerbe GO, Petty TL. Reinforcing breath carbon monoxide reductions in chronic obstructive pulmonary disease. *Drug & Alcohol Dependence* 1991; 29(1):47-62.
- (86) Halpern MT, Gillespie BW, Warner KE. Patterns of Absolute Risk of Lung Cancer Mortality in Former Smokers. *J Natl Cancer Inst* 1993; 85(6):457-464.
- (87) Peto R, Darby S, Deo H, Silcocks P, Whitley E, Doll R. Smoking, smoking cessation, and lung cancer in the UK since 1950: combination of national statistics with two case-control studies. *BMJ* 2000; 321(7257):323-329.
- (88) Ebbert JO, Yang P, Vachon CM, Vierkant RA, Cerhan JR, Folsom AR et al. Lung Cancer Risk Reduction After Smoking Cessation: Observations From a Prospective Cohort of Women. *J Clin Oncol* 2003; 21(5):921-926.
- (89) Borland R, Yong HH, Cummings KM, Hyland A, Anderson S, Fong GT et al. Determinants and consequences of smoke-free homes: findings from the International Tobacco Control (ITC) Four Country Survey. *Tob Control* 2006; 15 Suppl 3:iii42-iii50.
- (90) Holden C. 'Behavioral' Addictions: Do They Exist? *Science* 2001; 294(5544):980-982.
- (91) Zickler P. Nicotine's Multiple Effects on the Brain's Reward System Drive Addiction. NIDA [2003 [cited 2008 Apr. 19]; 17(6) Available from: URL:http://www.drugabuse.gov/NIDA_notes/NNVol17N6/Nicotine.html
- (92) West R. *Theory of Addiction*. first ed. Blackwell Publishing; 2006.
- (93) Lingford-Hughes AR, Welch S, Nutt DJ. Evidence-Based Guidelines for the Pharmacological Management of Substance Misuse, Addiction and Comorbidity: Recommendations from the British Association for Psychopharmacology. *J Psychopharmacol* 2004; 18(3):293-335.
- (94) Skog. Addicts' choice. *Addiction* 2000; 95(9):1309-1314.

- (95) Butler CC, Pill R, Stott NCH. Qualitative study of patients' perceptions of doctors' advice to quit smoking: implications for opportunistic health promotion. *BMJ* 1998; 316(7148):1878-1881.
- (96) STOTT NCH, PILL RM. 'Advise Yes, Dictate No'. Patients' Views on Health Promotion in the Consultation. *Fam Pract* 1990; 7(2):125-131.
- (97) Schofield I, Kerr S, Tolson D, Schofield I, Kerr S, Tolson D. An exploration of the smoking-related health beliefs of older people with chronic obstructive pulmonary disease. *Journal of Clinical Nursing* 2007; 16(9):1726-1735.
- (98) Copeland L. An exploration of the problems faced by young women living in disadvantaged circumstances if they want to give up smoking: can more be done at general practice level? *Fam Pract* 2003; 20(4):393-400.
- (99) Bancroft A, Wiltshire S, Parry O, Amos A. "It's like an addiction first thing... afterwards it's like a habit": daily smoking behaviour among people living in areas of deprivation. *Social Science & Medicine* 2003; 56(6):1261-1267.
- (100) Wiltshire S, Bancroft A, Parry O, Amos A. 'I came back here and started smoking again': perceptions and experiences of quitting among disadvantaged smokers. *Health Educ Res* 2003; 18(3):292-303.
- (101) Parrott AC. Does cigarette smoking cause stress? *American Psychologist* 1999; 54(10):817-820.
- (102) Bush J, White M, Kai J, Rankin J, Bhopal R. Understanding influences on smoking in Bangladeshi and Pakistani adults: community based, qualitative study. *BMJ* 2003; 326(7396):962.
- (103) Parry O, Thomson C, Fowkes G. Cultural context, older age and smoking in Scotland: qualitative interviews with older smokers with arterial disease. *Health Promot Int* 2002; 17(4):309-316.
- (104) Phillips R, Amos A, Ritchie D, Cunningham-Burley S, Martin C. Smoking in the home after the smoke-free legislation in Scotland: qualitative study. *BMJ* 2007; 335(7619):553.
- (105) Stead M, MacAskill S, MacKintosh AM, Reece J, Eadie D. "It's as if you're locked in": qualitative explanations for area effects on smoking in disadvantaged communities. *Health & Place* 2001; 7(4):333-343.
- (106) Poland BD, Cohen JE, Ashley MJ, Adlaf E, Ferrence R, Pederson LL et al. Heterogeneity among smokers and non-smokers in attitudes and behaviour regarding smoking and smoking restrictions. *Tob Control* 2000; 9(4):364-371.
- (107) Sutton S. How ordinary people in Great Britain perceive the health risks of smoking. *J Epidemiol Community Health* 1998; 52(5):338-339.

- (108) Roddy E, Antoniak M, Britton J, Molyneux A, Lewis S, Roddy E et al. Barriers and motivators to gaining access to smoking cessation services amongst deprived smokers--a qualitative study. *BMC Health Services Research* 2006; 6:147.
- (109) Wiltshire S, Bancroft A, Amos A, Parry O. "They're doing people a service"--qualitative study of smoking, smuggling, and social deprivation. *BMJ* 2001; 323(7306):203-207.
- (110) White M, Bush J, Kai J, Bhopal R, Rankin J. Quitting smoking and experience of smoking cessation interventions among UK Bangladeshi and Pakistani adults: the views of community members and health professionals. *J Epidemiol Community Health* 2006; 60(5):405-411.
- (111) Baillie AJ, Mattick RP, Hall W. Quitting smoking: estimation by meta-analysis of the rate of unaided smoking cessation. *Australian Journal of Public Health* 1995; 19(2):129-131.
- (112) Campbell IA. Smoking cessation. [Review] [47 refs]. *Thorax* 2000; 55 Suppl 1:S28-S31.
- (113) White AR, Rampes H, Campbell JL. Acupuncture and related interventions for smoking cessation. White AR, Rampes H, Campbell JL Acupuncture and related interventions for smoking cessation The Cochrane Database of Systematic Reviews: Reviews 2006 Issue 1 John Wiley & Sons, Ltd Chichester, UK DOI : 10 1002 /14651858 CD000009 pub2 2006.
- (114) Hughes JR, Stead LF, Lancaster T. Antidepressants for smoking cessation. Hughes JR, Stead LF, Lancaster T Antidepressants for smoking cessation The Cochrane Database of Systematic Reviews: Reviews 2004 Issue 4 John Wiley & Sons, Ltd Chichester, UK DOI : 10 1002 /14651858 CD000031 pub2 2004.
- (115) Bize R, Burnand B, Mueller Y, Cornuz J. Biomedical risk assessment as an aid for smoking cessation. [Review] [61 refs]. *Cochrane Database of Systematic Reviews* 2005;CD004705.
- (116) Secker-Walker RH, Gnich W, Platt S, Lancaster T. Community interventions for reducing smoking among adults. Secker Walker RH, Gnich W, Platt S, Lancaster T Community interventions for reducing smoking among adults The Cochrane Database of Systematic Reviews: Reviews 2002 Issue 3 John Wiley & Sons, Ltd Chichester, UK DOI : 10 1002 /14651858 CD001745 2002.
- (117) Sinclair HK, Bond CM, Stead LF. Community pharmacy personnel interventions for smoking cessation. Sinclair HK, Bond CM, Stead LF Community pharmacy personnel interventions for smoking cessation The Cochrane Database of Systematic Reviews: Reviews 2004 Issue 1 John Wiley & Sons, Ltd Chichester, UK DOI : 10 1002 /14651858 CD003698 pub2 2004.

- (118) Park EW, Schultz JK, Tudiver F, Campbell T, Becker L. Enhancing partner support to improve smoking cessation. Park E W , Schultz JK , Tudiver F , Campbell T , Becker L Enhancing partner support to improve smoking cessation The Cochrane Database of Systematic Reviews: Reviews 2004 Issue 3 John Wiley & Sons , Ltd Chichester, UK DOI : 10 1002 /14651858 CD002928 pu 2004.
- (119) Ussher M. Exercise interventions for smoking cessation. Ussher M Exercise interventions for smoking cessation The Cochrane Database of Systematic Reviews: Reviews 2005 Issue 1 John Wiley & Sons , Ltd Chichester, UK DOI : 10 1002 /14651858 CD002295 pub2 2005.
- (120) Stead LF, Lancaster T. Group behaviour therapy programmes for smoking cessation. Stead LF, Lancaster T Group behaviour therapy programmes for smoking cessation The Cochrane Database of Systematic Reviews: Reviews 2005 Issue 2 John Wiley & Sons , Ltd Chichester, UK DOI : 10 1002 /14651858 CD001007 pub2 2005.
- (121) Lancaster T, Stead LF. Individual behavioural counselling for smoking cessation. Lancaster T , Stead LF Individual behavioural counselling for smoking cessation The Cochrane Database of Systematic Reviews: Reviews 2005 Issue 2 John Wiley & Sons , Ltd Chichester, UK DOI : 10 1002 /14651858 CD001292 pub2 2005.
- (122) Silagy C, Lancaster T, Stead L, Mant D, Fowler G. Nicotine replacement therapy for smoking cessation. Silagy C , Lancaster T , Stead L , Mant D , Fowler G Nicotine replacement therapy for smoking cessation The Cochrane Database of Systematic Reviews: Reviews 2004 Issue 3 John Wiley & Sons , Ltd Chichester, UK DOI : 10 1002 /14651858 CD000146 pub2 2004.
- (123) Rice VH, Stead LF. Nursing interventions for smoking cessation. Rice VH , Stead LF Nursing interventions for smoking cessation The Cochrane Database of Systematic Reviews: Reviews 2004 Issue 1 John Wiley & Sons , Ltd Chichester, UK DOI : 10 1002 /14651858 CD001188 pub2 2004.
- (124) Lancaster T, Stead LF. Physician advice for smoking cessation. Lancaster T , Stead LF Physician advice for smoking cessation The Cochrane Database of Systematic Reviews: Reviews 2004 Issue 4 John Wiley & Sons , Ltd Chichester, UK DOI : 10 1002 /14651858 CD000165 pub2 2004.
- (125) Hajek P, Stead LF, West R, Jarvis M, Lancaster T. Relapse prevention interventions for smoking cessation. Hajek P , Stead LF, West R , Jarvis M , Lancaster T Relapse prevention interventions for smoking cessation The Cochrane Database of Systematic Reviews: Reviews 2005 Issue 1 John Wiley & Sons , Ltd Chichester, UK DOI : 10 1002 /14651858 CD003999 pub2 2005.

- (126) Lancaster T, Stead LF. Self-help interventions for smoking cessation. Lancaster T , Stead LF Self help interventions for smoking cessation The Cochrane Database of Systematic Reviews: Reviews 2005 Issue 3 John Wiley & Sons , Ltd Chichester, UK DOI : 10 1002 /14651858 CD001118 pub2 2005.
- (127) Abbot NC, Stead LF, White AR, Barnes J. Hypnotherapy for smoking cessation. Abbot NC , Stead LF, White AR, Barnes J Hypnotherapy for smoking cessation The Cochrane Database of Systematic Reviews: Reviews 1998 Issue 2 John Wiley & Sons , Ltd Chichester, UK DOI : 10 1002 /14651858 CD001008 1998.
- (128) Malcolm RE, Sillett RW, Turner JA, Ball KP. The Use of Nicotine Chewing Gum as an Aid to Stopping Smoking. *Psychopharmacology* 1980; 70:295-296.
- (129) Daughton D, Susman J, Sitorius M, Belenky S, Millatmal T, Nowak R et al. Transdermal Nicotine Therapy and Primary Care: Importance of Counseling, Demographic, and Participant Selection Factors on 1-Year Quit Rates. *Arch Fam Med* 1998; 7(5):425-430.
- (130) ICRF. Randomised trial of nicotine patches in general practice: results at one year. *BMJ* 1994; 308(6942):1476-1477.
- (131) Russell MA, Stapleton JA, Feyerabend C, Wiseman SM, Gustavsson G, Sawe U et al. Targeting heavy smokers in general practice: randomised controlled trial of transdermal nicotine patches. *BMJ* 1993; 306(6888):1308-1312.
- (132) Stapleton JA, Russell MA. Dose effects and predictors of outcome in a randomized trial of transdermal nicotine patches in general practice. *Addiction* 1995; 90(1):31-42.
- (133) Garvey AJ, Kinnunen T, Nordstrom BL, Utman CH, Doherty K, Rosner B et al. Effects of nicotine gum dose by level of nicotine dependence. *Nicotine & Tobacco Research* 2000; 2(1):53-63.
- (134) Hand S, Edwards S, Campbell IA, Cannings R. Controlled trial of three weeks nicotine replacement treatment in hospital patients also given advice and support. *Thorax* 2002; 57(8):715-718.
- (135) Yudkin P, Hey K, Roberts S, Welch S, Murphy M, Walton R. Abstinence from smoking eight years after participation in randomised controlled trial of nicotine patch. *BMJ* 2003; 327(7405):28-29.
- (136) Eisenberg MJ, Filion KB, Yavin D, Belisle P, Mottillo S, Joseph L et al. Pharmacotherapies for smoking cessation: a meta-analysis of randomized controlled trials. *CMAJ* 2008; 179(2):135-144.

- (137) NICE. Guidance on the use of nicotine replacement therapy (NRT) and bupropion for smoking cessation. NICE [2005 [cited 2008 July 14]; Available from:
URL:<http://www.nice.org.uk/nicemedia/pdf/NiceNRT39GUIDANCE.pdf>
- (138) Wagena EJ, van der Meer RM, Ostelo RJ, Jacobs JE, Van Schayck CP. The efficacy of smoking cessation strategies in people with chronic obstructive pulmonary disease: results from a systematic review. [Review] [37 refs]. *Respiratory Medicine* 2004; 98(9):805-815.
- (139) Brandt CJ, Ellegaard H, Joensen M, Kallan FV, Sorknaes AD, Tougaard L. Effect of diagnosis of "smoker's lung". *The Lancet* 1997; 349(9047):253.
- (140) Anthonisen NR, Connett JE, Kiley JP, Altose MD, Bailey WC, Buist AS et al. Effects of smoking intervention and the use of an inhaled anticholinergic bronchodilator on the rate of decline of FEV1. The Lung Health Study.[see comment]. *JAMA* 1994; 272(19):1497-1505.
- (141) Stead LF, Perera R, Bullen C, Mant D, Lancaster T. Nicotine replacement therapy for smoking cessation. Stead Lindsay F , Perera Rafael , Bullen Chris , Mant David , Lancaster Tim Nicotine replacement therapy for smoking cessation Cochrane Database of Systematic Reviews : Reviews 2008 Issue 1 John Wiley & Sons , Ltd Chichester, UK DOI : 10 1002 /14651858 2008.
- (142) Miller R, Rollnick S. *Motivational Interviewing*. Second ed. Guilford Press; 2002.
- (143) Burke BL, Hal Arkowitz, Christopher Dunn. *Efficacy of Motivational Interviewing and Its Adaptations*. The Gulliford Press; 2002. 217-250.
- (144) Greenland P, Gidding SS, Tracy RP. Commentary: Lifelong prevention of atherosclerosis: the critical importance of major risk factor exposures. *Int J Epidemiol* 2002; 31(6):1129-1134.
- (145) Ockene JK, Kuller LH, Svendsen KH, Meilahn E. The relationship of smoking cessation to coronary heart disease and lung cancer in the Multiple Risk Factor Intervention Trial (MRFIT). *Am J Public Health* 1990; 80(8):954-958.
- (146) Rollnick S, Butler CC, Stott N. Helping smokers make decisions: the enhancement of brief intervention for general medical practice. *Patient Education & Counseling* 1997; 31(3):191-203.
- (147) McClure JB. Are biomarkers a useful aid in smoking cessation? A review and analysis of the literature. *Behavioral Medicine* 2001; 27(1):37-47.

- (148) Walker WB, Franzini LR. Low-risk aversive group treatments, physiological feedback, and booster sessions for smoking cessation. *Behavior Therapy* 1985;(1985, vol. 16, no. 3, p. 263-274, ISSN: 0005-7894. Publisher: Assn for the Advancement of Behavior Therapy, US, <http://www.aabt.org.>)
- (149) Li VC, Kim YJ, Ewart CK, Terry PB, Cuthie JC, Wood J et al. Effects of physician counseling on the smoking behavior of asbestos-exposed workers. *Preventive Medicine* 1984; 13(5):462-476.
- (150) Greenhalgh T, Greenhalgh T. Papers that summarise other papers (systematic reviews and meta-analyses). [Review] [21 refs]. *BMJ* 1997; 315(7109):672-675.
- (151) Segnan N, Ponti A, Battista RN, Senore C, Rosso S, Shapiro SH et al. A randomized trial of smoking cessation interventions in general practice in Italy. *Cancer Causes & Control* 1991; 2(4):239-246.
- (152) Jamrozik K, Vessey M, Fowler G, Wald N, Parker G, Van VH. Controlled trial of three different antismoking interventions in general practice. *British Medical Journal Clinical Research Ed* 1984; . 288(6429):1499-1503.
- (153) Sanders D, Fowler G, Mant D, Fuller A, Jones L, Marzillier J. Randomized controlled trial of anti-smoking advice by nurses in general practice. *Journal of the Royal College of General Practitioners* 1989; 39(324):273-276.
- (154) Bovet P, Perret F, Cornuz J, Quilindo J, Paccaud F. Improved smoking cessation in smokers given ultrasound photographs of their own atherosclerotic plaques. *Preventive Medicine* 2002; 34(2):215-220.
- (155) Sippel JM, Osborne ML, Bjornson W, Goldberg B, Buist AS. Smoking cessation in primary care clinics.[comment]. *Journal of General Internal Medicine* 1999; 14(11):670-676.
- (156) Etter JF, Perneger TV. Effectiveness of a Computer-Tailored Smoking Cessation Program: A Randomized Trial. *Arch Intern Med* 2001; 161(21):2596-2601.
- (157) Bednarek M, Gorecka D, Wielgomas J, Czajkowska-Malinowska M, Regula J, Mieszko-Filipczyk G et al. Smokers with airway obstruction are more likely to quit smoking.[see comment]. *Thorax* 2006; 61:869-873.
- (158) Stratelis G, Molstad S, Jakobsson P, Zetterstrom O, Stratelis G, Molstad S et al. The impact of repeated spirometry and smoking cessation advice on smokers with mild COPD. *Scand J Prim Health Care* 2006; 24(3):133-139.
- (159) Buffels J, Degryse J, Decramer M, Heyrman J, Buffels J, Degryse J et al. Spirometry and smoking cessation advice in general practice: A randomised clinical trial. *Respiratory Medicine* 2006; 100(11):2012-2017.

- (160) Alderson P, Alderson P. The importance of theories in health care. [Review] [13 refs]. BMJ 1998; 317(7164):1007-1010.
- (161) Sutton S. Predicting Health Behaviour: Stage theories of health behaviour. In: Conner M, Norman P, editors. Predicting Health Behaviour. 2nd ed. Open University Press; 2005. 227.
- (162) Ogden J. Health beliefs. In: Ogden J, editor. 4 ed. Open University Press; 2007. 13-23.
- (163) Bandura. Self-efficacy: Toward a unifying theory of behavioral change. Psychological Review 1977; 84(2):191-215.
- (164) Meehl PE. Theoretical risks and tabular asterisks: Sir Karl, Sir Ronald, and the slow progress of soft psychology. [Article]. Journal of Consulting & Clinical Psychology 1978; 46(4):806-834.
- (165) Ogden J. Some Problems With Social Cognition Models: A Pragmatic and Conceptual Analysis. [Report]. Health Psychology 2003; 22(4):424-428.
- (166) Sutton S. Predicting and Explaining Intentions and Behavior: How Well Are We Doing? Journal of Applied Social Psychology 1998; 28(15):1317-1338.
- (167) Sutton S. Predicting and Explaining Intentions and Behavior: How Well Are We Doing? Journal of Applied Social Psychology 1998; 28(15):1317-1338.
- (168) Armitage CJ, Conner M, Armitage CJ, Conner M. Efficacy of the Theory of Planned Behaviour: a meta-analytic review. British Journal of Social Psychology 2001; 40(Pt 4):471-499.
- (169) Abraham C, Sheeran P. Predicting Health Behaviour: The Health belief model. In: Conner M, Norman P, editors. Predicting Health Behaviour. 2nd ed. Open University Press; 2005. 28.
- (170) Cahill K, Green N. Stage-based interventions for smoking cessation. Cochrane Database of Systematic Reviews 2007 [2008 [cited 2008 Feb. 15]; Available from:
URL: <http://mrw.interscience.wiley.com/cochrane/clsysrev/articles/CD004492/frame.html>
- (171) Fishbein M & Ajzen I. *Belief, Attitude, Intention, and Behavior: An Introduction to Theory and Research*. Reading, MA: Addison-Wesley.; 1975.
- (172) Schifter DE, Ajzen I, Schifter DE, Ajzen I. Intention, perceived control, and weight loss: an application of the theory of planned behavior. Journal of Personality & Social Psychology 1985; 49(3):843-851.
- (173) Miller K. *Communications theories: perspectives, processes, and contexts*. New York: McGraw-Hill.; 2005.

- (174) Godin G, Valois P, Lepage L, Desharnais R. Predictors of smoking behaviour: an application of Ajzen's theory of planned behaviour. *British Journal of Addiction* 1992; 87(9):1335-1343.
- (175) Christopher J. The theory of planned behavior and binge drinking: Assessing the impact of binge drinker prototypes. *Addictive Behaviors* 2007; 32(9):1753-1768.
- (176) Christopher J. Evidence that theory of planned behaviour variables mediate the effects of socio-demographic variables on homeless people's participation in service programmes. *Journal of Health Psychology* 2007; 12(5):805-817.
- (177) Desrichard ORSBL. The theory of planned behavior as mediator of the effect of parental supervision: A study of intentions to violate driving rules in a representative sample of adolescents. *Journal of Safety Research* 2007; 38(4):447-452.
- (178) Katherine M. Behavioral, normative and control beliefs underlying low-fat dietary and regular physical activity behaviors for adults diagnosed with type 2 diabetes and/or cardiovascular disease. *Psychology, health & medicine* 2007; 12(4):485-494.
- (179) Lautenschlager LSC. Understanding gardening and dietary habits among youth garden program participants using the Theory of Planned Behavior. *Appetite* 2007; 49(1):122-130.
- (180) Myklestad IRJ. Predicting willingness to engage in unsafe sex and intention to perform sexual protective behaviors among adolescents. *Health education & behavior* 2007; 34(4):686-699.
- (181) Ajzen I. Questions Raised by a Reasoned Action Approach: Comment on Ogden (2003).[Editorial]. *Health Psychology* 2004; 23(4):431-434.
- (182) Sutton. A Comparative Test of the Theory of Reasoned Action and the Theory of Planned Behavior in the Prediction of Condom Use Intentions in a National Sample of English Young People. *Health Psychology* 1999; 18(1):72-81.
- (183) Sparks P, Guthrie CA. Self-Identity and the Theory of Planned Behavior: A Useful Addition or an Unhelpful Artifice?1. *Journal of Applied Social Psychology* 1998; 28(15):1393-1410.
- (184) Hardeman W, Johnston M, Johnston D, Bonetti D, Wareham N, Kinmonth AL. Application of the Theory of Planned Behaviour in Behaviour Change Interventions: A Systematic Review. *Psychology & Health* 2002; 17(2):123-158.
- (185) Sheppard BH, Hartwick J, Warshaw PR. The Theory of Reasoned Action: A Meta-Analysis of Past Research with Recommendations for Modifications and Future Research. *The Journal of Consumer Research* 1988; 15(3):325-343.

- (186) Hale JLHBJ&GKL. The theory of reasoned action. . In: J.P.Dillard & M.Pfau (Eds.), editor. *The persuasion handbook: Developments in theory and practice*. Thousand Oaks, CA: Sage; 2003. 259-286.
- (187) Black DR, Babrow AS, Black DR, Babrow AS. Identification of campaign recruitment strategies for a stepped smoking cessation intervention for a college campus. *Health Education Quarterly* 1991; 18(2):235-247.
- (188) Coleman T, Barrett S, Wynn A, Wilson A. Comparison of the smoking behaviour and attitudes of smokers who believe they have smoking-related problems with those who do not. *Fam Pract* 2003; 20(5):520-523.
- (189) West R, Sohal T, West R, Sohal T. "Catastrophic" pathways to smoking cessation: findings from national survey. *BMJ* 2006; 332(7539):458-460.
- (190) Janz NK, Becker MH, Janz NK, Becker MH. The Health Belief Model: a decade later. [Review] [58 refs]. *Health Education Quarterly* 1984; 11(1):1-47.
- (191) Harrison JA, Mullen PD, Green LW. A meta-analysis of studies of the Health Belief Model with adults. *Health Educ Res* 1992; 7(1):107-116.
- (192) Maiman LA, Becker MH, Kirscht JP, Haefner DP, Drachman RH, Maiman LA et al. Scales for measuring health belief model dimensions: a test of predictive value, internal consistency, and relationships among beliefs. *Health Education Monographs* 1977; 5(3):215-230.
- (193) Kirscht JP, Haefner DP, Eveland JD, Kirscht JP, Haefner DP, Eveland JD. Public response to various written appeals to participate in health screening. *Public Health Reports* 1975; 90(6):539-543.
- (194) Haefner DP, Kirscht JP, Haefner DP, Kirscht JP. Motivational and behavioral effects of modifying health beliefs. *Public Health Reports* 1970; 85(6):478-484.
- (195) Bandura A, Adams NE, Beyer J, Bandura A, Adams NE, Beyer J. Cognitive processes mediating behavioral change. *Journal of Personality & Social Psychology* 1977; 35(3):125-139.
- (196) Kerr S, Watson H, Tolson D, Lough M, Brown M, Kerr S et al. Smoking after the age of 65 years: a qualitative exploration of older current and former smokers' views on smoking, stopping smoking, and smoking cessation resources and services. *Health & Social Care in the Community* 2006; 14(6):572-582.
- (197) Schmitz JM, Spiga R, Rhoades HM, Fuentes F, Grabowski J, Schmitz JM et al. Smoking cessation in women with cardiac risk: a comparative study of two theoretically based therapies. *Nicotine & Tobacco Research* 1999; 1(1):87-94.

- (198) Manfredi C, Lacey LP, Warnecke R, Petraitis J, Manfredi C, Lacey LP et al. Sociopsychological correlates of motivation to quit smoking among low-SES African American women. *Health education & behavior* 1998; 25(3):304-318.
- (199) Conrad KM, Campbell RT, Edington DW, Faust HS, Vilnius D, Conrad KM et al. The worksite environment as a cue to smoking reduction. *Research in Nursing & Health* 1996; 19(1):21-31.
- (200) Marshall P, Marshall P. "Just one more. . .!" A study into the smoking attitudes and behaviour of patients following first myocardial infarction. *International Journal of Nursing Studies* 1990; 27(4):375-387.
- (201) Kaufert JM, Rabkin SW, Syrotuik J, Boyko E, Shane F, Kaufert JM et al. Health beliefs as predictors of success of alternate modalities of smoking cessation: results of a controlled trial. *Journal of Behavioral Medicine* 1986; 9(5):475-489.
- (202) Stewart PJ, Rosser WW, Stewart PJ, Rosser WW. The impact of routine advice on smoking cessation from family physicians. *CMAJ* 1982; 126(9):1051-1054.
- (203) Giannetti VJ, Reynolds J, Rihn T. Factors which differentiate smokers from ex-smokers among cardiovascular patients: A discriminant analysis. *Social Science & Medicine* 1985; 20(3):241-245.
- (204) Li C, Unger JB, Schuster D, Rohrbach LA, Howard-Pitney B, Norman G. Youths' exposure to environmental tobacco smoke (ETS): Associations with health beliefs and social pressure. *Addictive Behaviors* 2003; 28(1):39-53.
- (205) Pederson LL, Baskerville JC, Wanklin JM. Multivariate statistical models for predicting change in smoking behavior following physician advice to quit smoking. *Preventive Medicine* 1982; 11(5):536-549.
- (206) Stacy D, Brenda H. An investigation of beliefs about smoking among diabetes patients: Information for improving cessation efforts. *Patient Education and Counseling* 1990; 15(2):181-189.
- (207) Weinberger M, Greene JY, Mamlin JJ, Jerin MJ. Health beliefs and smoking behavior. *Am J Public Health* 1981; 71(11):1253-1255.
- (208) Mullen PD, Hersey JC, Iverson DC. Health behavior models compared. *Social Science & Medicine* 1987; 24(11):973-981.
- (209) Prochaska JO, DiClemente CC. Stages and processes of self-change of smoking: toward an integrative model of change. *Journal of Consulting & Clinical Psychology* 1983; 51:390-395.

- (210) DiClemente CC, Prochaska JO, Fairhurst SK, Velicer WF, Velasquez MM, Rossi JS. The process of smoking cessation: an analysis of precontemplation, contemplation, and preparation stages of change. *Journal of Consulting & Clinical Psychology* 1991; 59(2):295-304.
- (211) Prochaska JO, Goldstein MG. Process of smoking cessation. Implications for clinicians. *Clinics in Chest Medicine* 1991; 12(4):727-735.
- (212) Sutton. Back to the drawing board? A review of applications of the transtheoretical model to substance use. *Addiction* 2001; 96(1):175-186.
- (213) Velicer WF, Rossi JS, DiClemente CC, Prochaska JO. A criterion measurement model for health behavior change. *Addictive Behaviors* 1996; 21(5):555-584.
- (214) Larabie LC. To what extent do smokers plan quit attempts? *Tob Control* 2005; 14(6):425-428.
- (215) Weinstein. Stage Theories of Health Behavior: Conceptual and Methodological Issues. *Health Psychology* 1998; 17(3):290-299.
- (216) Shadel WG, Shiffman S. Assessment of Smoking Behaviour. In: Donovan Dennis M, Marlatt G. Alan, editors. *Assessment of Addictive Behaviors*. 2nd ed. 2005. 113-146.
- (217) Audrain J, Boyd NR, Roth J, Main D, Caporaso NF, Lerman C. Genetic susceptibility testing in smoking-cessation treatment: one-year outcomes of a randomized trial. *Addictive Behaviors* 1997; 22(6):741-751.
- (218) Lerman C. Incorporating Biomarkers of Exposure and Genetic Susceptibility Into Smoking Cessation Treatment: Effects on Smoking-Related Cognitions, Emotions, and Behavior Change.[Article]. *Health Psychology* 1997; 16(1):87-99.
- (219) Glass TA, McAtee MJ. Behavioral science at the crossroads in public health: Extending horizons, envisioning the future. *Social Science & Medicine* 2006; 62(7):1650-1671.
- (220) Rigotti NA, Munafo MR, Murphy MFG, Stead LF. Interventions for smoking cessation in hospitalised patients. Rigotti NA , Munafo MR, Murphy MFG , Stead LF Interventions for smoking cessation in hospitalised patients *The Cochrane Database of Systematic Reviews: Reviews* 2002 Issue 4 John Wiley & Sons , Ltd Chichester, UK DOI : 10 1002 /14651858 CD001837 2002.
- (221) van Sluijs EMF, van Poppel MNM, van Mechelen W. Stage-based lifestyle interventions in primary care: Are they effective? *American Journal of Preventive Medicine* 2004; 26(4):330-343.

- (222) Pieterse ME, Seydel ER, DeVries H, Mudde AN, Kok GJ. Effectiveness of a Minimal Contact Smoking Cessation Program for Dutch General Practitioners: A Randomized Controlled Trial. *Preventive Medicine* 2001; 32(2):182-190.
- (223) Deakin MA, Deakin MA. Catastrophe modelling in the biological sciences. [Review] [135 refs]. *Acta Biotheoretica* 1990; 38(1):3-22.
- (224) Resnicow K, Vaughan R. A chaotic view of behavior change: a quantum leap for health promotion. *International Journal of Behavioral Nutrition and Physical Activity* 2006; 3(1):25.
- (225) Gladwell Malcolm. *The Tipping Point: How Little Things Can Make a Big Difference*. Little Brown; 2000.
- (226) Hufford MR, Witkiewitz K, Shields AL, Kodya S, Caruso JC, Hufford MR et al. Relapse as a nonlinear dynamic system: application to patients with alcohol use disorders. *Journal of Abnormal Psychology* 2003; 112(2):219-227.
- (227) Srinivasan H, Srinivasan H. Models for leprosy. An appraisal of graphic representations of the "spectrum" concept as models and a suggestion for a catastrophe theory model for leprosy. *International Journal of Leprosy & Other Mycobacterial Diseases* 1984; 52(3):402-413.
- (228) Abrams JH, Barke RA, Cerra FB, Abrams JH, Barke RA, Cerra FB. Quantitative evaluation of clinical course in surgical ICU patients: the data conform to catastrophe theory. *Journal of Trauma-Injury Infection & Critical Care* 1984; 24(12):1028-1037.
- (229) Clair SD. *An empirical test of a cusp catastrophe model for adolescent risky behavior*. US: ProQuest Information and Learning; 1997.
- (230) Byrne DG, Mazanov J, Gregson R-AM. *A cusp catastrophe analysis of changes to adolescent smoking behaviour in response to smoking prevention programs*. US: Society for Chaos Theory in Psychology and Life Sciences; 2001.
- (231) West R, West R. Time for a change: putting the Transtheoretical (Stages of Change) Model to rest.[see comment]. *Addiction* 2005; 100(8):1036-1039.
- (232) Trochim W2. *Social research methods. The Research Methods Knowledge Base*. 2nd Edition ed. Cincinnati, OH.: Atomic Dog Publishing, 2006.
- (233) Oppenheim AN. *Questionnaire Design, Interviewing and Attitude Measurement*. New Edition ed. London: Continuum; 1992.

- (234) Strecher VJ, Kreuter M, Den Boer DJ, Kobrin S, Hospers HJ, Skinner CS et al. The effects of computer-tailored smoking cessation messages in family practice settings.[see comment]. *Journal of Family Practice* 1994; 39(3):262-270.
- (235) Glasgow RE, Mullooly JP, Vogt TM, Stevens VJ, Lichtenstein E, Hollis JF et al. Biochemical validation of smoking status: pros, cons, and data from four low-intensity intervention trials. *Addictive Behaviors* 1993; 18(5):511-527.
- (236) Aaron SD, Vandemheen KL, Fergusson D, Maltais F, Bourbeau J, Goldstein R et al. Tiotropium in Combination with Placebo, Salmeterol, or Fluticasone Salmeterol for Treatment of Chronic Obstructive Pulmonary Disease: A Randomized Trial. *Ann Intern Med* 2007; 146(8):545-555.
- (237) Davis RA MSJdJR. Dietary nicotine: a source of urinary cotinine. *Food Chemistry & Toxicology* 2008; 29:821-827.
- (238) Jones PW. Health status measurement in chronic obstructive pulmonary disease. *Thorax* 2001; 56(11):880-887.
- (239) Department of Health. Statistics on smoking cessation services in England, April 2002 to March 2003. on line [2003 [cited 2008 Oct. 6]; Available from:
URL:http://www.dh.gov.uk/en/Publicationsandstatistics/Pressreleases/DH_4047481
- (240) Robert West. Assessing smoking cessation performance in NHS Stop Smoking Services: The Russell Standard (Clinical). Internet [2005 Available from:
URL:<http://www.aspsilverbackwebsites.co.uk/RobertWest/resources/russellstandardclinicalversion2.pdf>
- (241) micromedical.
http://www.micromedical.co.uk/products/proddetail2.asp?spiro_id=5.
2006.
Ref Type: Catalog
- (242) Kozlowski LT, Porter CQ, Orleans CT, Pope MA, Heatherton T, Kozlowski LT et al. Predicting smoking cessation with self-reported measures of nicotine dependence: FTQ, FTND, and HSI. *Drug & Alcohol Dependence* 1994; 34(3):211-216.
- (243) Fagerstrom KO. Measuring degree of physical dependence to tobacco smoking with reference to individualization of treatment. *Addictive Behaviors* 1978; 3(3-4):235-241.
- (244) Fagerstrom KO, Schneider NG, Fagerstrom KO, Schneider NG. Measuring nicotine dependence: a review of the Fagerstrom Tolerance Questionnaire. [Review] [65 refs]. *Journal of Behavioral Medicine* 1989; 12(2):159-182.

- (245) Hughes JR, Hatsukami D, Hughes JR, Hatsukami D. Signs and symptoms of tobacco withdrawal. Archives of General Psychiatry 1986; 43(3):289-294.
- (246) Blondal T, Blondal T. Controlled trial of nicotine polacrilex gum with supportive measures. Arch Intern Med 1989; 149(8):1818-1821.
- (247) Heatherton TF, KLF, RFKO. The Fagerstrom Test for Nicotine Dependence: a revision of the Fagerstrom Tolerance Questionnaire. British Journal of Addiction 1991; 86((9)):1119-1127.
- (248) Rios-Bedoya CF, Snedecor SM, Pomerleau CS, Pomerleau OF. Association of withdrawal features with nicotine dependence as measured by the Fagerstrom Test for Nicotine Dependence (FTND). Addictive Behaviors 2008; 33(8):1086-1089.
- (249) Moolchan ET, Radzins A, Epstein DH, Uhl G, Gorelick DA, Cadet JL et al. The Fagerstrom Test for Nicotine Dependence and the Diagnostic Interview Schedule: Do they diagnose the same smokers? Addictive Behaviors 2002; 27(1):101-113.
- (250) Shiffman S, West R, Gilbert D, SRNT Work Group on the Assessment of Craving and Withdrawal in Clinical Trials., Shiffman S, West R et al. Recommendation for the assessment of tobacco craving and withdrawal in smoking cessation trials.[see comment]. [Review] [96 refs]. Nicotine & Tobacco Research 2004; 6(4):599-614.
- (251) Persaud R. Motivated Mind. Bantam Books Ltd; New Ed edition (2 Jan 2006); 2006.
- (252) Maslow A. Motivation and Personality. Longman Asia Ltd ,Hong Kong; 3Rev Ed edition (31 Dec 1987); 1987.
- (253) Maslow AH. A theory of human motivation.[Article]. Psychological Review 1943; 50(4):370-396.
- (254) Motivation Theories - Cognitive Dissonance Theory. Internet [2008 [cited 2008 July 11]; Available from: URL:http://www.internet-of-the-mind.com/motivation_theories.html
- (255) Rollnick S, Mason P, Butler C. Health Behavior Change. Edinburgh London New York: Churchill Livingstone; 1999.
- (256) Edwards G, Lader M. Addiction: Processes of Change. Oxford University Press; 1994.
- (257) Biener L, Abrams DB, Biener L, Abrams DB. The Contemplation Ladder: validation of a measure of readiness to consider smoking cessation. Health Psychology 1991; 10(5):360-365.

- (258) Yong HH, Borland R, Siahpush M. Quitting-related beliefs, intentions, and motivations of older smokers in four countries: findings from the international tobacco control policy evaluation survey. *Addictive Behaviors* 2005; 30(4):777-788.
- (259) Hughes JR, Keely JP, Fagerstrom KO, Callas PW. Intentions to quit smoking change over short periods of time. *Addictive Behaviors* 2005; 30(4):653-662.
- (260) Richmond R, Webster I, Richmond R, Webster I. Evaluation of general practitioners' use of a smoking intervention programme. *Int J Epidemiol* 1985; 14(3):396-401.
- (261) Kotz D, Huibers MJ, West R, Wesseling G, Van-Schayck CP. What mediates confrontational counselling on smoking cessation in smokers with COPD? *Medical Hypotheses*. In press 2008.
- (262) Bandura A. Self efficacy: toward a unifying theory of behavioral change. *Psychol Rev* 1977; **84:191--215**.
- (263) Dijkstra A, de VH, Bakker M, Dijkstra A, de Vries H, Bakker M. Pros and cons of quitting, self-efficacy, and the stages of change in smoking cessation. *Journal of Consulting & Clinical Psychology* 1996; 64(4):758-763.
- (264) West R, McEwen A, Bolling K, Owen L. Smoking cessation and smoking patterns in the general population: a 1-year follow-up. *Addiction* 2001; 96(6):891-902.
- (265) Ockene JK, Emmons KM, Mermelstein RJ, Perkins KA, Bonollo DS, Voorhees CC et al. Relapse and maintenance issues for smoking cessation. [Review] [120 refs]. *Health Psychology* 2000; 19(1 Suppl):17-31.
- (266) Rimer BK, Orleans CT, Fleisher L, Cristinzio S, Resch N, Telepchak J et al. Does tailoring matter? The impact of a tailored guide on ratings and short-term smoking-related outcomes for older smokers.[see comment]. *Health Educ Res* 1994; 9(1):69-84.
- (267) Condiotte MM, Lichtenstein E, Condiotte MM, Lichtenstein E. Self-efficacy and relapse in smoking cessation programs. *Journal of Consulting & Clinical Psychology* 1981; 49(5):648-658.
- (268) Gwaltney CJ, Shiffman S, Norman GJ, Paty JA, Kassel JD, Gnys M et al. Does smoking abstinence self-efficacy vary across situations? Identifying context-specificity within the Relapse Situation Efficacy Questionnaire. *Journal of Consulting & Clinical Psychology* 2001; 69(3):516-527.
- (269) Norman P, Conner M, Bell R, Norman P, Conner M, Bell R. The theory of planned behavior and smoking cessation. *Health Psychology* 1999; 18(1):89-94.

- (270) Kotz D, Huibers MJ, Vos R, Van Schayck CP, Wesseling G, Kotz D et al. Principles of confrontational counselling in smokers with chronic obstructive pulmonary disease (COPD). *Medical Hypotheses* 2008; 70(2):384-386.
- (271) Peretti-Watel P, Constance J, Guilbert P, Gautier A, Beck F, Moatti JP. Smoking too few cigarettes to be at risk? Smokers' perceptions of risk and risk denial, a French survey. *Tob Control* 2007; 16(5):351-356.
- (272) Jones PW, Quirk FH, Baveystock CM. Why quality of life measures should be used in the treatment of patients with respiratory illness. *Monaldi Archives for Chest Disease* 1994; 49(1):79-82.
- (273) Spencer S, Calverley PM, Sherwood BP, Jones PW, ISOLDE Study Group. Health status deterioration in patients with chronic obstructive pulmonary disease. *American Journal of Respiratory & Critical Care Medicine* 2001; 163(1):122-128.
- (274) Calverley PM, Boonsawat W, Cseke Z, Zhong N, Peterson S, Olsson H. Maintenance therapy with budesonide and formoterol in chronic obstructive pulmonary disease. *Eur Respir J* 2003; 22(6): 912-919:-919.
- (275) Bouchet C, Guillemin F, Hoang-Thi TH, Cornette A, Briancon S. Validity of a disease specific quality of life instrument, the St George's Respiratory Questionnaire, in patients with chronic respiratory disease. *Revue des Maladies Respiratoires* 1996; 13(1): 43-46:-46.
- (276) Calverley PMA. Minimal clinically important difference - Exacerbations of COPD. *COPD: Journal of Chronic Obstructive Pulmonary Disease* 2005; 2(1): 143-148:-148.
- (277) Brusasco V, Hodder R, Miravittles M, Korducki L, Towse L, Kesten S. Health outcomes following treatment for six months with once daily tiotropium compared with twice daily salmeterol in patients with COPD. *Thorax* 2003; 58(5): 399-404:-404.
- (278) Guyatt GH, Berman LB, Townsend M, Pugsley SO, Chambers LW. A measure of quality of life for clinical trials in chronic lung disease. *Thorax* 1987; 42(10):773-778.
- (279) Barley EA, Quirk FH, Jones PW. Asthma health status measurement in clinical practice: validity of a new short and simple instrument. *Respiratory Medicine* 1998; 92(10):1207-1214.
- (280) HAJIRO TAKA, NISHIMURA KOIC, JONES PAUL, TSUKINO MITS, IKEDA AKIH, KOYAMA HIRO et al. A Novel, Short, and Simple Questionnaire to Measure Health-related Quality of Life in Patients with Chronic Obstructive Pulmonary Disease. *Am J Respir Crit Care Med* 1999; 159(6):1874-1878.
- (281) Jones PW, Quirk FH, Baveystock CM. The St George's Respiratory Questionnaire. *Respiratory Medicine* 1991; 85 Suppl B:25-31.

- (282) Jones PW, Quirk FH, Baveystock CM, Littlejohns P. A self-complete measure of health status for chronic airflow limitation. The St. George's Respiratory Questionnaire. *American Review of Respiratory Disease* 1992; 145(6):1321-1327.
- (283) Keogh-Brown MR, Bachmann MO, Shepstone L, Hewitt C, Howe A, Ramsay CR et al. Contamination in trials of educational interventions. [Review] [276 refs]. *Health Technology Assessment (Winchester, England)* 2007; 11(43):iii-107.
- (284) Adams G, Gulliford MC, Ukoumunne OC, Eldridge S, Chinn S, Campbell MJ. Patterns of intra-cluster correlation from primary care research to inform study design and analysis. *Journal of Clinical Epidemiology* 2004; 57(8):785-794.
- (285) Kerry SM, Bland JM. Statistics notes: Sample size in cluster randomisation. *BMJ* 1998; 316(7130):549.
- (286) Campbell MK, Elbourne DR, Altman DG. CONSORT statement: extension to cluster randomised trials. *BMJ* 2004; 328(7441):702-708.
- (287) National Institute of Health and Clinical Excellence. Smoking cessation: draft guidance. NICE [2003 Available from: URL:<http://guidance.nice.org.uk/page.aspx?o=429278>
- (288) Craig P, Dieppe P, Macintyre S, Michie S, Nazareth I, Petticrew M. Developing and evaluating complex interventions: the new Medical Research Council guidance. *BMJ* 2008; 337(sep29_1):a1655.
- (289) Stead LF, Lancaster T, Perera R. Telephone counselling for smoking cessation. Stead LF, Lancaster T , Perera R Telephone counselling for smoking cessation *The Cochrane Database of Systematic Reviews: Reviews* 2003 Issue 1 John Wiley & Sons , Ltd Chichester, UK DOI : 10 1002 /14651858 CD002850 2003.
- (290) Glassman AH, Helzer JE, Covey LS, Cottler LB, Stetner F, Tipp JE et al. Smoking, smoking cessation, and major depression. *JAMA: The Journal of the American Medical Association* 1990; 264(12):1546-1549.
- (291) Covey LS, Glassman AH, Stetner F. Major depression following smoking cessation. *Am J Psychiatry* 1997; 154(2):263-265.
- (292) Greenhalgh T, Taylor R. How to read a paper: Papers that go beyond numbers (qualitative research). *BMJ* 1997; 315(7110):740-743.
- (293) U.S.Preventive Services Task Force. Screening for Chronic Obstructive Pulmonary Disease Using Spirometry: U.S. Preventive Services Task Force Recommendation Statement. *Ann Intern Med* 2008; 148(7):529-534.

- (294) Department of Health. Best Research for Best Health: A new national health research strategy. Internet [2008 [cited 2008 Oct. 22]; Available from:
URL:http://www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsPolicyAndGuidance/DH_4127127
- (295) The L. Is primary-care research a lost cause? The Lancet 2003; 361(9362):977.
- (296) Sheikh A, Smeeth L, Ashcroft R, Sheikh A, Smeeth L, Ashcroft R. Randomised controlled trials in primary care: scope and application. [Review] [74 refs]. British Journal of General Practice 2002; 52(482):746-751.
- (297) McManus RJ, Ryan R, Jones M, Wilson S, Hobbs FR. How representative of primary care are research active practices? Cross-sectional survey. Fam Pract 2008; 25(1):56-62.
- (298) Macfarlane F, Shaw S, Greenhalgh T, Carter YH. General practices as emergent research organizations: a qualitative study into organizational development. Fam Pract 2005; 22(3):298-304.
- (299) Van Schayck CP, Chavannes NH. Detection of asthma and chronic obstructive pulmonary disease in primary care. [Review] [50 refs]. European Respiratory Journal - Supplement 2003; 39:16s-22s.
- (300) U.S.Preventive Services Task Force. Screening for Chronic Obstructive Pulmonary Disease Using Spirometry: U.S. Preventive Services Task Force Recommendation Statement. Ann Intern Med 2008; 148(7):529-534.
- (301) Qaseem A, Snow V, Shekelle P, Sherif K, Wilt TJ, Weinberger S et al. Diagnosis and Management of Stable Chronic Obstructive Pulmonary Disease: A Clinical Practice Guideline from the American College of Physicians. Ann Intern Med 2007; 147(9):633-638.
- (302) Smith-sivertsen T, Rortveit G. Should general practitioners screen smokers for COPD? Scand J Prim Health Care 2004; 22(4):196-201.
- (303) Department of Health. UK NATIONAL SCREENING COMMITTEE. Internet [2008 [cited 2008 Nov. 13]; Available from:
URL:<http://www.nsc.nhs.uk/>
- (304) White P, Wong W, Fleming T, Gray B. Primary care spirometry: test quality and the feasibility and usefulness of specialist reporting. The British Journal of General Practice 2008; 57:701-705.
- (305) Schermer TR, Jacobs JE, Chavannes NH, Hartman J, Folgering HT, Bottema BJ et al. Validity of spirometric testing in a general practice population of patients with chronic obstructive pulmonary disease (COPD). Thorax 2003; 58(10):861-866.

- (306) Quanjer PH, Tammeling GJ, Cotes JE, Pedersen OF, Peslin R, Yernault JC et al. Lung volumes and forced ventilatory flows. Report Working Party Standardization of Lung Function Tests, European Community for Steel and Coal. Official Statement of the European Respiratory Society.[see comment]. [Review] [290 refs]. European Respiratory Journal - Supplement 1993; 16:5-40.
- (307) Badgett RG, Tanaka DJ. Is Screening for Chronic Obstructive Pulmonary Disease Justified? Preventive Medicine 1997; 26(4):466-472.
- (308) Tashkin DP, Celli B, Senn S, Burkhart D, Kesten S, Menjoge S et al. A 4-Year Trial of Tiotropium in Chronic Obstructive Pulmonary Disease. N Engl J Med 2008; 359(15):1543-1554.
- (309) Wilt TJ, Niewoehner D, MacDonald R, Kane RL. Management of Stable Chronic Obstructive Pulmonary Disease: A Systematic Review for a Clinical Practice Guideline. Ann Intern Med 2007; 147(9):639-653.
- (310) British Lung Foundation. Brown's screening programme overlooks UK's second biggest killer. Internet [2008 [cited 2008 Oct. 11]; Available from: URL:<http://www.lunguk.org/media-and-campaigning/media-centre/archive-press-releases-and-statements/jan2008/BrownsscreeningprogrammeoverlooksUKssecondbiggestkiller.htm>
- (311) Glasziou P, Irwig L, Mant D. Monitoring in chronic disease: a rational approach. BMJ 2005; 330(7492):644-648.
- (312) Gordis L. Bias ,Confounding, and Interaction. Epidemiology. Second ed. Philadelphia: W.B.Saunders Company; 2000.
- (313) Schermer TRJ, Smeele IJM, Thoonen BPA, Lucas AEM, Grootens JG, van Boxem TJ et al. Current clinical guideline definitions of airflow obstruction and COPD overdiagnosis in primary care. Eur Respir J 2008; 32(4):945-952.
- (314) Prochaska JJ, Hall SM, Tsoh JY, Eisendrath S, Rossi JS, Redding CA et al. Treating Tobacco Dependence in Clinically Depressed Smokers: Effect of Smoking Cessation on Mental Health Functioning. Am J Public Health 2008; 98(3):446-448.

11 Appendices

11.1 Appendix 1. Glossary

Spirometry

A subject is asked to blow into a tube connected to a machine (spirometer), which can measure the different patterns of airflow and the rate and amount of air that is expelled from the lungs. The information can help to decide if there is damage to the lungs, and assist in distinguishing between different types of lung disease. Standard measurements include FEV1, FVC and the ratio FEV1/FVC (see below).

FEV1. Forced expiratory Volume 1.

Forced expiratory Volume 1 is the amount of air expelled by the lungs in the first one second by forcibly breathing out measured with a spirometer. This is compared to normal tables for height, age, gender and racial origin to allow expression as percentage of 'normal'.

FVC.

This is the total amount of air that can be expelled from the lungs with one forced breath after a full breath in.

11.2 Appendix 2. Summary of Cochrane review 2005

Summary of studies included in the Cochrane review of biomedical markers.¹¹⁵

Bize R, Burnand B, Mueller Y, Cornuz J. Biomedical risk assessment as an aid for smoking cessation. [Review] [61 refs]. Cochrane Database of Systematic Reviews 2005

Three studies in the review tested the effect of exhaled carbon monoxide measurements,^{152;153;217} three tested the combination of exhaled carbon monoxide measurement and spirometry,^{36;148;155} and one tested the effect of spirometry alone.¹⁵¹ One study included in the review looked at the influence of showing pictures of arterial damage in individual smoker's ultrasound tests.¹⁵⁴ Another study investigated the effect of information about genetic susceptibility to lung cancer.²¹⁷

The trials were conducted in three different settings: five trials took place either in general practice¹⁵¹⁻¹⁵³ or in outpatient clinics,^{154;155} two in a 'smoking clinic'^{148;217}, and one in a health promotion clinic for army veterans.³⁶ Four trials took place in the United States,^{36;148;155;217} two in the United Kingdom,^{152;153} one in Italy¹⁵¹ and one in the Seychelles Islands.¹⁵⁴

The mean age of the participants, when given, varied between 35.5 years and 53.7 years. The proportion of females in the trials varied between 4% and 63%. The mean number of cigarettes smoked per day varied between 11.9 and 29.2. The mean number of cigarettes smoked per day was highest in the trials set in a 'smoking clinic' (35.5 per day in Walker 1985¹⁴⁸, 22.7 per day in Audrain 1997²¹⁷) or among veterans.³⁶

Levels of nicotine addiction as assessed by the Fagerström score and

proportions of patients in the various stages of change according to Prochaska and Di Clemente were only given in some trials and could not be used to compare the study populations of the different trials.

Three of the trials isolated the effect of exhaled CO on smoking cessation rates resulting in the following odds ratios and 95% confidence intervals (95% CI): 0.73 (0.38 to 1.39), 0.93 (0.62 to 1.41), and 1.18 (0.84 to 1.64). Combining CO measurement with genetic susceptibility gave an OR of 0.58 (0.29 to 1.19).

Exhaled CO measurement and spirometry were used together in three trials, resulting in the following ORs (95% CI): 0.6 (0.25 to 1.46), 2.45 (0.73 to 8.25), and 3.50 (0.88 to 13.92).

Spirometry results alone were used in one other trial with an OR of 1.21 (0.60 to 2.42).

Methodological problems

Only one of the eight trials reported an adequate randomisation procedure.¹⁵¹ Four studies did not report the method of randomisation, or did not give enough information to assume that allocation was adequately concealed.^{36;148;154;217} Three reported inadequate concealment of allocation, with allocation by day or week of attendance,^{152;153} or by odd/even numbered questionnaire at the time of check-in.¹⁵⁵

Remarkably, only one study included a power calculation estimation of sample size prior to recruitment.¹⁵⁵ This estimate was based on a quit rate of 25% in the intervention group versus 10% in the control group. This is probably a rather optimistic estimate of the possible effect of intervention.

All studies included male and female adults who were smokers at the time of inclusion. Only two studies gave a definition of being a smoker at the time of inclusion.^{154;217}

Urinary cotinine level was used to validate smoking cessation at follow up in two trials.^{151;153} One study used the same validation procedure but only on a sub-sample (41%) of self-reported ex-smokers.¹⁵² Two studies used expired air carbon monoxide.^{36;148} Two studies did not use any biochemical validation.^{154;155}

Methods of recruitment varied widely. Among the three studies conducted in general practice, one recruited patients at their first visit,¹⁵² another screened outpatients on specific days¹⁵¹ and the last screened all outpatients during the recruitment period.¹⁵³ One study recruited smokers among outpatients in primary care clinics.¹⁵⁵ The two studies conducted in 'smoking cessation clinics' recruited smokers by media advertisement.^{148;217} The remaining two studies recruited the last consecutive 155 smokers who participated in a health survey,¹⁵⁴ and veterans that responded to mailed invitation to attend a health promotion clinic.³⁶ Participation rates (i.e. the proportion of those approached who agreed to take part in the trial) were seldom recorded.

11.3 Appendix 3. Questionnaires

11.3.1 Beliefs about quitting

Some of the questions used to measure beliefs about quitting in the study by Hyland et al 'Individual-level predictors of cessation behaviours among participants in the International Tobacco Control (ITC) Four Country Survey'.⁸⁰ (see Section 3.2.1, 'Why do people start or stop smoking?')

Beliefs about quitting
Intention to quit In next month, in next 6 months, beyond 6 months, not planning to quit: This variable comes from the question 'Are you planning to quit smoking (in next month, in next 6 months, beyond 6 months, not planning to quit)?'
Self-efficacy of quitting Current smokers were asked, 'If you decided to give up smoking completely in the next 6 months, how sure are you that you would succeed?' Categories of the answer include: not at all sure, slightly sure, moderately sure, very sure, and extremely sure. The variable was coded as a continuous measure from 1 (weak) to 5 (strong).
Smokers were asked, 'How much do you think you would benefit from health and other gains if you were to quit smoking permanently in the next 6 months?' Categories of the answer include: not at all, slightly, moderately, very much, and extremely. The variable was coded as a continuous measure from 1 (weak) to 5 (strong) .

Worries about health and quality of life

This variable was created based on smokers' responses to two questions at baseline:

- (1) 'How worried are you, if at all, that smoking will lower your quality of life in the future?'
- (2) 'How worried are you, if at all, that smoking will damage your health in the future?'

Categories of the answers include: not at all worried, a little worried, moderately worried, very worried.

Each variable was coded as a continuous measure from 1 (weak) to 4. (strong) and the average of the two measures was used in analyses

Favourable attitudes about smoking

This variable was created based on smokers' response to the following two statements:

- (1) 'You enjoy smoking too much to give it up';
- (2) 'Smoking is an important part of your life'.

Each variable was coded as a continuous measure from 1 (weak) to 5 (strong) and the average of the two measures is used in analyses.

Overall attitude about smoking

At baseline, smokers were asked: 'What is your overall opinion of smoking?' The variable was coded into a three-category variable: positive, neutral or negative.

11.3.2 Airways Questionnaire 20/30

(The AQ20 comprises the first 20 items. A 'Yes' response is scored '1', 'No' and 'N/A' responses are scored '0'.)

The following - questions are concerned with the effect of your chest trouble on your everyday life. Please respond 'Yes', 'No' or 'Not Applicable' (N/A) to each item.

- (1) Do you suffer from coughing attacks during the day?
- (2) Because of your chest trouble do you often feel restless?
- (3) Because of your chest trouble do you feel breathless maintaining the garden?
- (4) Do you worry when going to a friend's house that there might be something there that will set off an attack of chest trouble?
- (5) Do you suffer from chest symptoms as a result of exposure to strong smells, cigarette smoke or perfume?
- (6) Is your partner bothered by your chest trouble?
- (7) Do you feel breathless while trying to sleep?
- (8) Do you worry about the long-term effects on your health of the drugs that you have to take because of your chest trouble?
- (9) Does getting emotionally upset make your chest trouble worse?
- (10) Because of your chest trouble are there times when you have difficulty getting around the house?
- (11) Because of your chest trouble do you suffer from breathlessness carrying out activities at work?
- (12) Do you feel breathless walking upstairs because of your chest trouble?
- (13) Because of your chest trouble do you suffer from breathlessness doing housework?

- (14) Because of your chest trouble do you go home sooner than others after a night out?
- (15) Because of your chest trouble do you suffer from breathlessness when you laugh?
- (16) Because of your chest trouble do you often feel impatient?
- (17) Because of your chest trouble do you feel that you cannot enjoy a full life?
- (18) Do you feel drained after a cold because of your chest trouble?
- (19) Do you have a feeling of chest heaviness?
- (20) Do you bother much about your chest trouble?
- (21) Do you have difficulty taking part in sports because of your chest trouble?
- (22) Do you worry about getting an attack of chest trouble even when you are well?
- (23) Are you embarrassed by heavy breathing?
- (24) Does your chest trouble affect you other than when you are having an attack?
- (25) Do you do all the things you want to regardless of the effects on your chest trouble?
- (26) Because of your chest trouble do you often feel helpless?
- (27) Do you work badly when your chest trouble is bad?
- (28) Because of your chest trouble do you have difficulty doing housework?
- (29) Is your sex life affected by your chest trouble?
- (30) Do you suffer from discomfort when you cough?

11.3.3 St. George's Respiratory Questionnaire (SGRQ)

This questionnaire is designed to help us learn much more about how your breathing is troubling you and how it affects your life. We are using it to find out which aspects of your illness cause you most problems rather than what the doctors and nurses think your problems are.

Please read the instructions carefully and as if you do not understand anything. Do not spend too long deciding about your answer

Before completing the rest of the questionnaire:

Please tick one box to show how you describe your current health:

Very good	
Good	
Fair	
Poor	
Very poor	

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P.W. Jones PhD FRCP

Professor of Respiratory Medicine

St George's Hospital Medical School

Jenner Wing

Cranmer Terrace

London SW17 0RE

Tel. 020 8725 5371

Part 1. St. George's Respiratory Questionnaire

Questions about how much chest trouble you have had over the past 4 weeks.

Please tick (✓) one box for each question

Over the past 4 weeks,	Most days a week	Several Days a week	A few days a month	Only with Chest infection	Not at All
1. I have coughed:					
2. I have brought up phlegm (sputum)					
3. I have had shortness of breath					
4. I have had attacks of wheezing					

5. During the past 4 weeks, how many severe or very unpleasant attacks of chest trouble you have had?	More than 3 attacks	
	3 attacks	
	2 attacks	
	1 attack	
	No attacks	

6. During the past 4 weeks, how long did the worst attack of chest trouble last?	More than 3 attacks	
	A week or more	
	3 or more days	
	1 or 2 days	
	Less than a day	

7. Over past 4 weeks, in an average week, how many	No good days	
--	--------------	--

good days (with little chest trouble) have you had?		
	1 or 2 good days	
	3 or 4 good days	
	Nearly every day	
	Every day is good	

8. If you have wheeze, is it worse in the morning?	No	
	Yes	

Part 2. St. George's Respiratory Questionnaire

Section 1

How would you describe your chest condition? Please tick (✓) one:

The most important problem I have	
Causes me quite a lot of problems	
Causes me a few problems	
Causes no problem	

If you have ever had paid employment. Please tick (✓) one:

My chest trouble made me stop work together	
My chest trouble interferes with my work or made me change my work	
My chest trouble does not affect my work	

Section 2

Questions about what activities usually make you feel breathless these days.

Please tick (✓) in each box that applies to you these days:

	True	False
Sitting or lying still		
Getting washed or dressed		
Walking around the home		
Walking outside on the level		
Walking up a flight of stairs		
Walking up hills		
Playing sports or games		

Part 2. St. George's Respiratory Questionnaire

Section 3

Some more questions about your cough and breathlessness these days

Please tick (✓) in each box that applies to you these days:

	True	False
My cough hurts		
My cough makes me tired		
I am breathless when I talk		
I am breathless when I bend over		
My cough or breathing disturbs my sleep		
I get exhausted easily		

Section 4

Questions about how your chest trouble may have on you these days.

Please tick (✓) in each box that applies to you these days:

	True	False
My cough or breathing is embarrassing in public		
My chest trouble is a nuisance to my family, friends or neighbours		
I get afraid or panic when I cannot get my breath		
I feel that I am not in control of my chest problem		
I do not expect my chest to get any better		
I have become frail or and invalid because of my chest		
Exercise is not safe for me		
Everything seems too much of an effort		

Section 5

Questions about your medication, if you are receiving no medication tick here ()

And go to Section 6.

Please tick (✓) in each box that applies to you these days:

	True	False
My medication does not help me very much		
I get embarrassed using my medication in public		
I have unpleasant side effects from my medication		
My medication interferes with my life a lot		

Part 2. St. George's Respiratory Questionnaire

Section 6

These are questions about how your activities might be affected by your breathing.

Please tick (✓) in each box that applies to you because of your breathing:

	True	False
I take a long time to get washed or dressed		
I cannot take a bath or shower, or take a long time		
I walk slower than other people, or I stop for a rest		
Jobs such as homework take a long time, or have to stop for rests		
If I walk up one flight of stairs, I have to stop or slow down		
My breathing makes it difficult to do things such as walk up hills, Carry things up stairs, garden, dance, play bowls or golf		
My breathing makes it difficult to do things such as carry heavy Loads, dig the garden or shovel snow, jog or walk at 5 miles per Hour, play tennis or swim		
My breathing makes it difficult to do things such as very heavy manual work, run, cycle, swim fast or play competitive sports		

Section 7

We would like to know your chest trouble usually affects your daily life.

Please tick (✓) in each box that applies to you these days:

	True	False
I cannot play sports or games		
I cannot go out for entertainment or recreation		
I cannot go out of the house to do the shopping		
I cannot do housework		
I cannot move far from my bed or chair		

Please now tick (✓) in the box (only one) which you think best describes how your chest affects you.

It does not stop me doing anything I would like to do	
It stops me doing one or two things I would like to do	
It stops me doing most of the things I would like to do	
It stops me doing everything I would like to do	

Thank you for filling in this questionnaire. Before you finish would you please check to see that you have answered all the questions.

11.3.4 Permission to use SGRQ

From: G P <parkesko@hotmail.co.uk>

Date: Mon, 27 Oct 2008 10:08:05 +0000

To: <pjones@sgul.ac.uk>

Subject: FW: Copy of questionnaire. Permission and publication

Dear Prof Jones

thank you for arranging for a copy of the SGRQ to be sent.

Sorry to be a nuisance for asking again.

Please could you confirm that you give permission for me to reproduce the St George's Respiratory Questionnaire in my PhD thesis?

Best wishes

Gary ParkesTo: parkesko@hotmail.co.uk

Subject: Re: Copy of questionnaire. Permission and publication

From: pjones@sgul.ac.uk

Date: Mon, 27 Oct 2008 12:11:17 +0000

Of course. Permission granted.

Paul J Sent from my BlackBerry® wireless device

11.4 Appendix 4. Stages of change correspondence

From: diclemen@umbc.edu

To: Gary Parkes

Subject: Using the three questions as an outcome measure?

These have been used extensively. Preparation are those who say yes to 30 days and to a quit attempt in the past year. If you are going to assess every 6 months, I would change the quit attempt time frame to 6 months rather than a year so the time frames do not overlap. Sorry for the delay. I have been travelling.

Carlo

11.5 Appendix 5. Letters to participants and GPs

11.5.1 Letters to participants

Practice Headed Paper

Dear <insert patient name>

Our practice is participating in a study of people over the age of 35 who smoke and we would like to invite you to take part. If you no longer smoke please let us know and we will amend your medical record.

Before you decide, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information and ask if anything is not clear or you would like more information. Thank you for reading this.

The purpose of the study

The study is looking at ways that smoking affects your health and is attempting to find a new way of motivating people to stop smoking.

Why you have been chosen

People who are smokers over the age of 35 years are being invited to take part. If you receive this by post your doctor has sent it out to you because he/she has a record in his notes that you are a smoker. The researchers will only know about you if you respond to this invitation and give consent to participate. Otherwise no one except your usual doctor(s) will know or have any access to your records.

We have enclosed a full information leaflet, which should answer most of your questions.

If you would like to take part please contact the practice on 01920<.....> or the research office on 01992 464900.

Someone from your Doctors' surgery will phone you in the next few weeks to offer you an appointment.

Yours sincerely

Dr..... and partners

11.5.2 Information sheet for participants

Information sheet about research study

Before you decide to be a volunteer for the test, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information and ask if anything is not clear or you would like more information. Thank you for reading this.

Frequently Asked Question

1. Who can volunteer for the test?

Anyone who is 35 years old or over AND is currently a smoker may be eligible. There are some people who are unsuitable, such as those with lung cancer or who have had part of a lung removed.

2. I have no intention of quitting smoking; can I still have the test?

Yes. The testing is about informing people about their own health and even if you decide to continue smoking you will be able to see the change in your lungs over the study period.

3. I am already thinking of (or trying to give up) giving up smoking can I still have the test?

Yes. Even if you are thinking of quitting you are still able to have the test. As long as you are still a smoker at the first visit for testing you can be included in the study.

4. Who cannot volunteer for the study?

Most people over 35 are eligible for the study even if they have other conditions like diabetes, heart disease, asthma, or chronic bronchitis. However there are some diseases, which prevent the test being done or make it unreliable. There is a full list of things that exclude people from the study on the 'questionnaire

page' If you are still unsure please discuss with the research assistant or contact the research administrator.

5. What is the purpose of the study?

The study is looking at ways that smoking affects your health and is attempting to find a new way of giving information about the health of your lungs and empowering people to stop smoking.

6. What happens if I change my mind?

Refusal to take part will not affect your normal care from your own doctor. If you decide to take part you can withdraw your consent at any time without needing to give an explanation.

7. What do I have to do?

The study does not involve any experimental drugs. Your medical care will continue to be in the control of you and your own doctor.

The study will involve you attending a designated testing site (usually a local GP surgery) as near to your home as possible on two occasions over a twelve month period. You will also be contacted about 3 to 6 months after the initial testing to check there are no questions or problems.

The research doctor or nurse will ask you about your past medical history and to fill in a questionnaire about your quality of life related to breathing function.

You will be asked to do a simple breathing test.

A random half of the participants will be given information about their lung function in a new way.

After repeating the test and a questionnaire in 12 months all participants will be told how their health and lungs have changed and a report will be given to their own doctor (with your written consent).

Everyone will be given standard basic information about stopping smoking.

8. How much time does it take?

The initial assessment takes between 30 and 40 minutes. The second visit will be shorter and take about 15 to 20 minutes.

9. What are the advantages of taking part?

After the results are complete you will be able to find out how smoking has affected your health and how it has changed over a 12-month period. If you give your written consent, at the end of the study your own doctor will get a report containing information, which is not routinely available.

10. Is it confidential?

When the results are analysed and/or published any information will be anonymous and any personal details will be strictly confidential to the researchers.

11. Do I get any payment for volunteering?

The study is funded by a research charity called The Health Foundation. No financial incentive can be offered although we hope to offer either a token or travel expenses to thank you for your time and participation. We are also planning to have prize draw towards the end of the study, and your name will be included, unless you opt out.

12. I live outside the area can I volunteer?

Unfortunately, the rules governing research mean that at the moment we can only test people in Hertfordshire and West Essex where ethical committee approval has already been obtained.

13. Any other questions please e-mail us on info@step2quit.co.uk

or telephone 01992 <.....> and speak to <.....> (9am-5pm or leave a message) or contact <.....name> at the <.....surgery>.

11.5.3 Post-test personalised letters to participants

Letter to intervention group

Address

Participant Number.

Date of examination:

Dear M.

We hope that you may benefit from knowing about the effect of smoking on your lung function. Your results are displayed below.

Your 'lung age' was measured as < > years compared with your actual age of < >.

LUNG AGE. There is a natural gradual decline in function of the lungs with age. Smoking can make your lungs decline more quickly as if the lungs are ageing more rapidly. The good news is that if you stop smoking the 'rate' of decline will return to normal.

If you have access to the internet you can see more detailed explanation on www.step2quit.co.uk

FEV1. Amount of air blown in 1 second	< >litres	Reversibility testing was done/not done Result positive / negative FEV1 changed < >% with 400mcg salbutamol
FVC.Total volume of lungs	< >litres	
Your LUNG AGE	< >years	

This type of lung function test does not tell us anything about the risk of other serious diseases related to smoking such as Lung cancer or Heart disease or Stroke. Smoking cessation is therefore still important for all people regardless of their age or the results of these lung tests. The test will be repeated in 12 months to measure any change.

Yours sincerely

Dr.

Letter to control group

Address

Participant Number

Date of examination:

Dear M

We hope that you and many others may benefit from more knowledge about the effect that smoking is having on your lung function. Your results are displayed below. An explanation of the terms used is below.

FEV1	< >litres	Reversibility testing was done/not done
FVC	< >litres	Result positive / negative

FEV1 changed < > % with 400mcg salbutamol.

They will be repeated after 12 months for comparison whether you are still smoking or not.

FEV1. This is the amount of air expelled by the lungs in the first one second by forcibly breathing out into a machine called a spirometer.

FVC. This is the total amount of air that can be expelled from the lungs with one forced breath after a full breath in.

The lung function test (Spirometry) will be done again in 12 months to measure any change due to your smoking.

This type of lung function test does not tell us anything about the risk of other serious diseases related to smoking such as Lung cancer or Heart disease or Stroke. Smoking cessation is therefore still important for all people regardless of their age or the results of these lung tests.

Thank you for taking part

Yours sincerely

Dr G Parkes.

For further help with smoking cessation contact your GP surgery or the local Hertfordshire Help line 0800 389 3998 or speak to your local pharmacist.

11.5.4 Letters for restrictive abnormality

Date of examination:

Re:

Address:

Dear M.

We hope that you may benefit from more knowledge about the effect that smoking is having on your lung function. After your visit and examination on <date> I have reviewed your results and looked at the information you gave to the researcher about your past medical history. Although I cannot be certain from one set of tests, the results suggest you may have a condition called a restrictive disorder as well as any changes from the smoking.

Because of this I am sending a copy of this letter to your GP (Dr<.....>[code]). It may be that there is already some record in your notes about this condition. Please make an appointment to see your GP who will want to review your record and decide if it is necessary for you to have some more investigations.

We will still invite you to return for a second examination in 12 months.

Thank you for taking part

Yours sincerely

Dr G Parkes.

For further help with smoking cessation contact your GP surgery or the local Hertfordshire Help line 0800 389 3998 or speak to your local pharmacist.

Date.		Litres	% of predicted value
Baseline results	FEV1		
	FVC		
Reversibility testing	FEV1		
% change	FVC		

cc. Dr. < >

Letter to general practitioner about restrictive abnormality

Dr.

Address

Dear Dr. < name > ,

Your patient, <name> has consented to take part in this research study investigating the value of screening for lung damage in smokers above the age of 35 years.

After their visit and examination on < date>, I have reviewed the results and looked at the information they gave to the research assistant about their past medical history. The results suggest they may have a restrictive disorder as well as any obstructive changes from the smoking. They have declared that they have a previous diagnosis of (deleted as per data) COPD/ asthma/other lung disease.

However, It may be that there is already some record in their notes to account for the restrictive spirometry changes, (e.g. intrinsic causes such as TB, fibrosis, drug induced interstitial disease, sarcoid; extrinsic causes such as musculo-skeletal disorders of the chest or spine).

If the past medical history does not reveal an adequate explanation for this finding, please arrange for any further clinical evaluation or investigation that may be necessary.

Your patient has consented to return for a second research examination in 12 months.

Yours sincerely

Dr G Parkes.

P.T.O. for results

Date.		Litres	% of predicted value	Comment
Baseline results	FEV1			>80% =normal
	FVC			>80% =normal
Reversibility testing	FEV1		% change	>15% =reversible
FEV1/FVC ratio	%			>70% normal
Assessment and recommendation	Normal/restrictive/Obstructive. Mixed restrictive and obstructive			Stop smoking Check records Investigate cause

Encl. spirometry

11.6 Appendix 6. Correspondence with NSF

(see Section 2.3.2)

From: parkesko@hotmail

To: COPD NSF@DOH.co.uk> cc:

Subject: New evidence for screening smokers 27/11/2007 18:26

I would like to bring to your attention new evidence to support the spirometric lung testing of all smokers aged over 35 I attach a copy of the RCT which is currently under peer review for publication by the BMJ. In summary:

1. Screening smokers (and ex- smokers) results in a large number of people with previously undiagnosed lung damage.
2. Giving spirometry results in the form of 'lung age' promotes smoking cessation. In fact this research reveals a doubling of successful smoking cessation

Yours faithfully

Dr G Parkes

From: COPD_NSF@dh.gsi.gov.uk

Subject: Re: New Evidence for Screening smokers

To: parkesko@hotmail.co.uk

Date: Tue, 18 Dec 2007 16:06:01 +0000

Dear Dr Parkes

Thank you very much for sending this article for our attention. I have forwarded to our evidence review team in order to ensure that this is included within our evidence on which the National Service Framework will be based.

Best wishes,

Fiona Phillips

COPD NSF Team

Department of Health

11.7 Appendix 7. Published research

BMJ

RESEARCH

Effect on smoking quit rate of telling patients their lung age: the Step2quit randomised controlled trial

Gary Parkes, general practitioner,¹ Trisha Greenhalgh, professor,² Mark Griffin, lecturer in medical statistics,² Richard Dent, consultant chest physician department of chest medicine³

¹The Limes Surgery, Hoddesdon, Hertfordshire EN11 8EP

²Department of Primary Care and Population Sciences, University College London, London N19 5LW

³Queen Elizabeth II Hospital, Welwyn Garden City, Hertfordshire AL7 4HQ

Correspondence to: G Parkes
Parkesko@hotmail.co.uk

doi:10.1136/bmj.39503.582396.25

ABSTRACT

Objective To evaluate the impact of telling patients their estimated spirometric lung age as an incentive to quit smoking.

Design Randomised controlled trial.

Setting Five general practices in Hertfordshire, England.

Participants 561 current smokers aged over 35.

Intervention All participants were offered spirometric assessment of lung function. Participants in intervention group received their results in terms of "lung age" (the age of the average healthy individual who would perform similar to them on spirometry). Those in the control group received a raw figure for forced expiratory volume at one second (FEV₁). Both groups were advised to quit and offered referral to local NHS smoking cessation services.

Main outcome measures The primary outcome measure was verified cessation of smoking by salivary cotinine testing 12 months after recruitment. Secondary outcomes were reported changes in daily consumption of cigarettes and identification of new diagnoses of chronic obstructive lung disease.

Results Follow-up was 89%. Independently verified quit rates at 12 months in the intervention and control groups, respectively, were 13.6% and 6.4% (difference 7.2%, $P=0.005$, 95% confidence interval 2.2% to 12.1%; number needed to treat 14). People with worse spirometric lung age were no more likely to have quit than those with normal lung age in either group. Cost per successful quitter was estimated at £280 (€366, \$556). A new diagnosis of obstructive lung disease was made in 17% in the intervention group and 14% in the control group; a total of 16% (89/561) of participants.

Conclusion Telling smokers their lung age significantly improves the likelihood of them quitting smoking, but the mechanism by which this intervention achieves its effect is unclear.

Trial registration National Research Register N0096173751.

INTRODUCTION

A quarter of smokers develop chronic obstructive pulmonary disease (COPD),¹ which is largely caused by smoking and is the fourth commonest cause of death worldwide.² In the United Kingdom, half of the estimated 1.5 million people with chronic obstructive

pulmonary disease are currently undiagnosed.³ According to the National Institute for Health and Clinical Excellence, the mean delay from onset to diagnosis is 20 years.⁴ Spirometry can detect obstructive lung damage in susceptible individuals after 20 pack years of smoking, typically at around age 35. Yet the average age of diagnosis of chronic obstructive pulmonary disease in the UK is 55, despite widespread availability of diagnostic equipment.⁴

Early diagnosis of chronic obstructive pulmonary disease with communication of lung damage to patients could improve targeting of smoking cessation programmes and improve quit rates in individuals most vulnerable to lung damage.⁵ A Cochrane review of the use of personal biomarkers (carbon monoxide measurements, spirometry, arterial damage) for the harmful effects of smoking, however, failed to find firm evidence that such markers could be used to increase the quit rate.⁶ A recent non-randomised observational study on the effect of communicating spirometry findings on smoking cessation concluded that "a large randomised clinical trial is needed to answer this important question more conclusively."⁷

The concept of "lung age" (the age of the average person who has an FEV₁ equal to the individual) was developed in 1985 as a way of making spirometry data easier to understand and also as a potential psychological tool to show smokers the apparent premature ageing of their lungs.⁵ We tested the hypothesis that telling smokers their lung age would lead to successful smoking cessation, especially in those with most damage.

METHOD

Management and governance

The research advisory group comprised a respiratory physician (RD), an academic general practitioner (TG), and the principal investigator (GP). A core management group, comprising principal investigator, practice manager, two practice nurses, healthcare assistant, and a patient representative, was responsible for the day to day running of the project.

Sampling and recruitment

A power calculation indicated the need for about 300 participants to have 80% power to detect a 10%

Table 1 | Stages of change questions (adapted from Prochaska¹⁴)

Stage	Descriptor	Defining question
Pre-contemplative	Not even thinking about changing	Answers "no" to the question "Are you intending to quit smoking in the next 6 months?"
Contemplative	Thinking about changing	Answers "yes" to previous question and "no" to the question "Are you intending to quit smoking in the next month?"
Preparation	Making plans to change	Answers "yes" to previous two questions and "no" to the question "Did you try to quit smoking in the past year?"
Action	Actively trying to change	Answers "yes" to the question "Did you try to quit smoking in the past year?"
Maintenance*	Having achieved change, is trying to maintain it	Answers "Yes" to the question "Have you given up smoking?"

*Question on "maintenance" phase used only in follow-up assessment as all participants were current smokers at baseline.

difference in smoking cessation rate (for example, 5% in one group v 15% in the other). Assuming an attrition rate of up to 50%, we aimed to recruit 600 participants. We searched computerised patient records from five general practices in Hertfordshire to identify people aged 35 and over who had been recorded as a smoker in the previous 12 months. We excluded those receiving oxygen and those with a history of lung cancer, tuberculosis, asbestosis, silicosis, bronchiectasis, or pneumonectomy. We sent a letter of invitation to participate in the study and a research information sheet. Two weeks later, we telephoned all those who had not already responded, offering an invitation to participate and to answer any queries. Those who could not be contacted by telephone were sent a second letter. Recruitment started in February 2004 and follow-up was completed in March 2007.

Assessment interview

All potential participants were asked to confirm that they were current smokers, had understood the information provided, and would be available for re-

assessment in 12 months. Baseline data included age, smoking history in pack years (average daily number of cigarettes smoked divided by 20 and multiplied by the number of years of smoking), medical history for exclusion criteria (see above), medication (especially use of steroids or antibiotics for chest infections in the preceding 12 months), and comorbidity including chronic bronchitis or emphysema, asthma, other lung disease, diabetes, treatment for blood pressure, stroke, coronary heart disease (angina or heart attack), or other heart disease. These comorbidities were not used as exclusion criteria but to confirm baseline comparability of groups.

All participants underwent standard measurements of lung function (FEV₁, FVC (forced vital capacity), FEV₁/FVC) with a Micromedical spirometer. Reversibility of airways obstruction was measured according to standard British Thoracic Society guidelines (over 15% and at least 400 ml improvement in FEV₁ after 400 µg salbutamol via a spacer).⁸ Both groups were told that their lung function would be measured again after 12 months to see whether it had deteriorated. They were not randomised until after spirometry had been completed. All participants were strongly encouraged to give up smoking and advised how to access local NHS smoking cessation clinics.

We used two instruments to confirm baseline comparability of groups: the St George's respiratory questionnaire and Prochaska's stages of change questions in relation to smoking. The St George's respiratory questionnaire is a validated questionnaire designed to be self administered under supervision and to measure the impact of respiratory diseases (in particular asthma and chronic obstructive pulmonary disease) on an individual's life.⁹ Like other quality of life instruments, it has the potential to identify a threshold response to therapy or compare the response to different therapies, or both.¹⁰ Scores of 7 or below indicate normal lung function. We adapted stage of change questions (with permission) from Prochaska and DiClemente's model in which smokers are asked three questions and classified on the basis of their response as in the "pre-contemplative," "contemplative," "preparation," or "action" phase (table 1).

Randomisation procedure

A clerk (who then took no further part in the study) prepared 600 sequentially numbered opaque sealed envelopes, each containing a card with allocation group determined by computer generated random number (odd = intervention). If the participant met the inclusion criteria and gave consent, he or she was entered into the study and underwent baseline spirometry. The next numbered envelope in the series was then opened to determine allocation group.

Instruments and tests

All data collectors were trained in the use of MicroLab 3500 spirometers (Micro Medical, Chatham, Kent), which were newly purchased at the start of the study. Spirometry readings were checked for internal

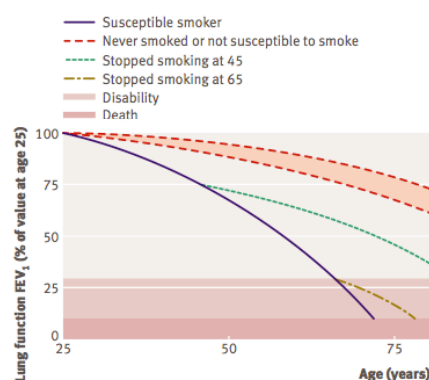


Fig 1 | Graph of lung function against age showing how smoking accelerates age related decline in lung function (adapted from Fletcher and Peto¹¹)

reliability on three criteria: at least two FEV₁ readings within 5% of each other; good quality time volume curve; and the internal spirometer computer display had to register "good blow." Smoking cessation at follow-up was initially assessed by measuring carbon monoxide concentrations with a Smoke Check SC01 monitor (Micro Medical, Chatham, Kent). This model has a carbon monoxide range of 0-500 ppm and a sensitivity of 1 ppm.

One of two independent nurses, who were blinded to allocation group, collected saliva samples for cotinine testing and recorded those who continued to take nicotine replacement therapy. Specimens were processed by ABS Laboratories, Medical Toxicology Unit, London. The optimum cut-off point to distinguish smokers from non-smokers is 14.2 ng/ml, which correctly classifies 99% of non-smokers and 96% of smokers. As the half life of cotinine is about 20 hours, the test would detect most people who had smoked a cigarette within the past 24-48 hours.

Estimation of lung age

Figure 1, adapted from the work of Fletcher and Peto, illustrates how smoking effectively "ages" the lungs.¹¹ The examples illustrated show how the lungs can deteriorate more rapidly with smoking, as if they are ageing faster. Smoking cessation will not allow the lungs to return to normal but reduction in function or "ageing" will then occur at a normal rate. Originally, calculation of lung age was based on estimates developed by Morris and Temple with reference linear regression equations to establish the best method.⁵ They showed that FEV₁ was the best test for calculating lung age mathematically (box). In practice, the lung age is automatically generated by adjustment of the settings of the spirometer.

Information given to participants

Participants in the intervention group were given their results verbally, immediately after randomisation, in the form of "lung age" with a graphic display (figs 1 and 2). The graphs were used as a visual aid to explain how the lung function normally reduces gradually with age and that smoking can damage lungs as if they are ageing more rapidly than normal. As an example a line can be drawn vertically up from the horizontal axis (fig 2) from "age 52" to reach the bold blue curve illustrating the lung function of the "susceptible smoker" and then horizontally to the curve representing those who have "never smoked" and lung function at age 75. Furthermore they were told that smoking cessation would slow

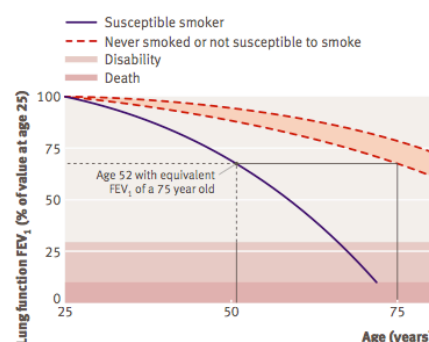


Fig 2 Explaining lung age to participants (adapted from Fletcher and Peto¹¹)

down the rate of deterioration of the lung function back to normal but would not repair the damage already done.

In the intervention group, if the lung age was equal to or less than the individual's chronological age, he or she was informed that test result was normal. If lung age was greater than chronological age, we gave them the "lung age" in years.

We did not tell those in the control group their results but informed them that they would be invited for a second test after 12 months to "see if there had been any change in lung function." If the examiner was pressed for more information, he or she could tell participants that they would receive a letter with more information from the research doctor within four weeks.

The principal research doctor (GP) reviewed all the results, checked the quality of the spirometry tracing, and considered the result in the light of clinical data. When there was doubt, he sent the results to a chest physician (RD) for interpretation and advice. Within four weeks of data collection the research doctor sent all participants an individualised letter. Written results were given to the control group as simple FEV₁ (litres per second) with no further explanation. Written results were given to the intervention group as "lung age."

The letter to both groups included the phrase "This type of lung function test does not tell us anything about the risk of other serious diseases related to smoking such as lung cancer or heart disease or stroke. Smoking cessation is therefore still important for all people regardless of their age or the results of these lung tests." All participants were given written contact details of the local NHS smoking cessation services.

In both groups, when reversibility testing indicated asthma (over 15% and at least 400 ml improvement in FEV₁ after 400 µg salbutamol via a spacer) we advised participants to attend their general practitioner for further management, and informed the general practitioner separately. When spirometry findings suggested restrictive lung disease, we sent the participant and his or her general practitioner a letter to alert them to the

Lung age calculation formula developed by Morris and Temple⁵

Men

Lung age = $2.87 \times \text{height (in inches)} - (31.25 \times \text{observed FEV}_1 \text{ (litres)}) - 39.375$

Women

Lung age = $3.56 \times \text{height (in inches)} - (40 \times \text{observed FEV}_1 \text{ (litres)}) - 77.28$

Table 2 | Baseline characteristics of groups. Figures are means (SDs) unless stated otherwise

	Control (n=281)	Intervention (n=280)
Age (years)	53 (11.9)	52.9 (11.9)
% (No) of men	47 (132)	45 (127)
Pack years	30.3 (19.3)	31.1 (17.7)
Daily cigarette consumption	17.4 (8.2)	16.5 (9)
Spirometry result:		
FEV ₁ % predicted	90 (19.8)	89 (19.8)
% FEV ₁ /FVC	75 (11.8)	73 (11.7)
% with abnormal FEV ₁ —that is, <80% of predicted (No)	23.5 (66)	26.8 (75)
SGRQ score	28.9 (22.4)	26.7 (22.0)
% (No) with medical history:		
COPD	7.2 (19)	7.7 (20)
Asthma	11 (29)	9.3 (24)
Other lung disease	2.7 (7)	2.3 (6)
CVA or stroke	4.2 (11)	0.8 (2)
CHD (angina or heart attack)	5.3 (14)	2.3 (6)
Other heart disease	2.3 (6)	1.2 (3)
Diabetes	5.7 (15)	3.5 (9)
Treatment for hypertension	21.3 (56)	19.1 (49)
% (No) with new diagnosis of COPD	17.4 (49)	14.3 (40)
% (No) at stage of change:		
No of patients*	263	260
Pre-contemplative	29.3 (77)	29.2 (76)
Contemplative	32.3 (85)	31.9 (83)
Preparation	16 (42)	18.1 (47)
Action	22.4 (59)	20.8 (54)

FEV₁, forced expiratory volume in one second; FVC=forced vital capacity; SGRQ=St George's respiratory questionnaire; CVA=cerebrovascular accident; CHD=coronary heart disease; COPD=chronic obstructive pulmonary disease.

*Not collected in pilot study, therefore data missing for 38 participants.

advisability of further investigation and guidelines on referral to secondary care.

Outcome measures

The primary outcome measure was verified cessation of smoking 12 months after the initial recruitment interview and examination. Secondary outcomes were changes in daily consumption of cigarettes and the identification of new diagnoses.

Follow-up and confirmation of cessation

Participants underwent follow-up examination with repeat spirometry after 12 months. Self reported quitters had carbon monoxide breath testing immediately for confirmation of smoking cessation, and they were informed that they would be contacted by an independent nurse for a saliva test for cotinine measurement.

Data analysis

We analysed data on an intention to treat basis and performed statistical analysis with SPSS version 11.0. We used unpaired *t* tests for continuous data and χ^2 tests for categorical data, except when expected cells were found to be less than 5, in which case we used Fisher's exact test.

To test the hypothesis that severity of lung damage predicts quit success, we used the *t* test to compare the

mean "lung age deficit" (difference of lung age minus chronological age) between quitters and non-quitters within the intervention group.

Assessment of costs

Though we did not carry out a full economic evaluation, we had accurate data on the time taken to carry out the spirometry tests and for results to be communicated to patients by letter. We calculated costs in terms of the time spent per patient processed and also per successful quitter.

RESULTS

Baseline characteristics

We recruited 561 participants (table 2). There were few significant differences between the groups at baseline, in particular groups did not differ in their quality of life score or stage of change. There were, however, significantly more people with a history of stroke in the control group. The incidence of comorbidity was high (around 20% of all participants), reflecting our deliberate intention not to exclude high risk individuals (and, perhaps, the inability or unwillingness of many smokers to quit despite the presence of considerable medical morbidity).

Despite an average of 33 pack years of smoking, most participants in this study had "normal" results on spirometry at baseline, which accords with previous studies on comparable populations.¹ According to British Thoracic Society cut-off values, only 23.5% of the control group and 26.8% of the intervention group had baseline lung function in the "abnormal" range.

Progress and outcome

Figure 3 shows progress through the trial and losses to follow-up. Table 3 shows follow-up data at 12 months. All recruited participants were included in the final data analysis. We analysed those who did not return for follow-up (32 and 31, respectively, in the control and intervention group) as if they continued to smoke. Verified quit rates were 6.4% (18/281) in the control group and 13.6% (38/280) in the intervention group (difference 7.2%, *P*=0.005, 95% confidence interval 2.2% to 12.1%). Telling participants their lung age was thus associated with an absolute reduction of 7.2% in the smoking rate compared with giving them their lung function tests results as raw FEV₁ data. The number needed to treat (NNT) for the intervention to achieve one additional sustained quitter is 14. Both groups reduced their average self reported consumption of cigarettes (table 3); average consumption at follow-up was significantly lower in the intervention group than in the control group (11.7 (SD 9.7) v 13.7 (SD 10.5) per day, *P*=0.03).

We recorded the numbers of smokers in both groups who used additional help to quit (health service clinics, nicotine replacement, bupropion, acupuncture, hypnosis): numbers were 22 (7.8%) in the control group and 30 (10.7%) in the intervention group (*P*=0.2).

Within the intervention group we investigated the effect of lung age deficit (lung age minus actual age) on

the likelihood of quitting. To investigate whether those with poorer lung function were more likely to quit we used independent samples *t* test to compare the mean deficit between those who were confirmed to have stopped smoking (*n*=38) versus the rest (*n*=242). The mean lung age deficit was 8.7 years and 9.4 years in the quitters and non-quitters, respectively. This difference was not significant (difference in means -0.78 , 95% confidence interval -7.6 to 6.0 , $P=0.8$). Thus, there was no evidence that those individuals with poorer lung age deficits were more likely to quit. The study was not powered to investigate this relation, however, and the lack of a significant result might be because of the small numbers, particularly in the quit group.

Costs

It took a healthcare assistant 30 minutes to perform a spirometry test. The principal investigator (GP) spent a further 15 minutes per patient reviewing results and preparing an individualised feedback letter, and this required about 10 minutes of secretarial and receptionist support. Using 2007 salary costs for the relevant staff, we estimate the cost of this intervention at £20 (£26, \$40) per patient processed and £280 (£366, \$556) per successful quitter (given a number needed to treat of 14).

DISCUSSION

This large randomised controlled trial with adequate follow-up and independent proof of cessation has shown that individualised feedback of "lung age" is effective in promoting smoking cessation. This study strongly supports the policy of giving patients their spirometry results expressed as "lung age" along with advice about the dangers of continuing to smoke and methods of quitting.

Comparison with other research

In 2001 a non-systematic overview analysed 12 studies that provided feedback on personal biomarkers as part of strategies to change behaviour in smokers.¹² The authors concluded that success was likely to depend on how the information was conveyed and understood and how it related to behaviour. They also suggested that success might depend on graphic displays or

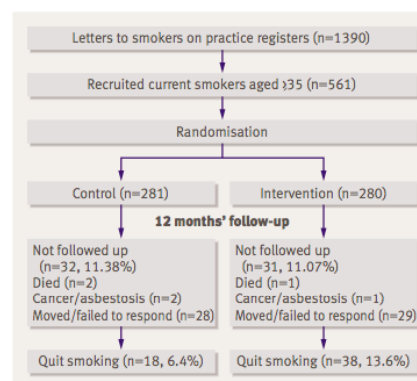


Fig 3 | Flow of participants through trial

written individualised information as well as the prospect of gain rather than negative messages about costs or disadvantage.

A Cochrane review of the evidence for the effectiveness of biomarkers in smoking cessation was published in October 2005.⁶ Observational studies were included in the background discussion but only randomised controlled trials were included in the analysis, which concluded that because of limited evidence no definitive statements could be made about the effectiveness of assessment of biomarkers as an aid for smoking cessation.⁶ None of the primary studies included in the Cochrane review had used "lung age" in the intervention. The negative conclusions of that review should be updated in the light of this new study.

The debate about the usefulness of screening with spirometry was recently rekindled by a large non-randomised observational study of 4494 smokers from Poland.⁷ Their results indicated that spirometry promoted cessation. Those with airways obstruction were more likely to quit, but even the group with normal lungs on spirometry had a higher quit rate (12%) than would normally be expected after simple advice from a physician (4-6%).¹³ They did not use "lung age" to explain results to participants but did use a visual display of Fletcher and Peto's diagram¹¹ to compare the participant's result with the average for age and project the likely deterioration with continued smoking. These authors did not have a control group but attributed the high quit rates in those with normal lung function to a "healthy volunteer" effect (those who had opted for the programme were seen as more motivated to quit).

The results of our study are broadly consistent with the findings of the Polish observational study, with one important difference. Contrary to the conclusions of the latter (and to clinical speculation), we found no evidence that successful quitting depends on the severity of lung damage as demonstrated by spirometry. Our study, however, was not powered to detect this difference, and we found, for example, that a 45 year

Table 3 | Results at 12 months. Figures are percentages (numbers) unless stated otherwise

	Control (n=281)	Intervention (n=280)	P value
Lost to follow-up	11.4 (32)	11.0 (31)	0.9
Smoking status			
Confirmed cessation*	6.4 (18)	13.6 (38)	0.01
Smoker at 12 months	90.4 (254)	84.6 (237)	
Unknown	3.2 (9)	1.8 (5)	
Mean (SD) daily cigarette consumption	13.7 (10.5)	11.7 (9.7)	0.03
Attended NHS smoking clinics	1.4 (4)	1.7 (5)	
Used smoking cessation help (clinic, NRT, bupropion, acupuncture)	7.8 (22)	10.7 (30)	0.2†

NRT=nicotine replacement therapy.

*Cotinine and CO measurement.

† χ^2 test.

WHAT IS ALREADY KNOWN ON THIS TOPIC

There is insufficient evidence to make a definitive statement about the evidence for the effectiveness of biomarkers (including spirometry) in smoking cessation

WHAT THIS STUDY ADDS

Smoking cessation rates can be improved by reporting estimation of lung age with spirometry in primary care

Screening smokers over the age of 35 could reduce smoking and improve early diagnosis of chronic obstructive pulmonary disease

old smoker who is told that their "lung age" is normal is as likely to quit as one who is told that his or her "lung age" is 65. Presentation of information in an understandable and visual way, whether the news is positive or negative, seems to encourage higher levels of successful smoking cessation than when patients are given feedback that is not easily understandable.

What makes people quit

What triggers the decision to quit and which methods result in successful and sustained quitting? Clinical experience suggests that deterioration in health does not necessarily lead to altered behaviour, whether that is related to smoking, drugs, or diet. The high rate of comorbidity (20%) in our participants confirms that many people who are likely to exacerbate a chronic health problem by smoking continue to smoke. Anecdotally, some participants in our trial were relieved when the results were found to be normal and therefore thought it was "not too late" to be trying to quit.

This apparent win-win situation might explain the apparently paradoxical finding that knowing one's lung age helps a smoker to quit whatever the result. If lung age is normal there is an incentive to stop before it is too late. If lung age is abnormal then this is a clear message that the lungs are undergoing accelerated deterioration that would be slowed if the smoker stopped. Further research is needed to elucidate the psychological forces that are active in successful quitting in different circumstances.

In this study, we measured stage of change (using Prochaska and DiClemente's transtheoretical model¹⁴) to ensure that the groups were comparable for this variable at baseline, but the study was underpowered to test the hypothesis that a smoker in the "active" phase of quitting would find feedback on lung age more useful than someone in the "pre-contemplative" phase. Some addiction experts have proposed that the transtheoretical model should be rejected in favour of a new integrated model.^{15 16} Any new psychological theory of smoking cessation will need to explain the unexpected finding that normal results within personal biomarkers are as likely to promote cessation as abnormal ones.

Current National Institute for Health and Clinical Excellence guidelines include one on brief interventions and referral for smoking cessation^{17 18} (which do not mention spirometry testing at all) and another on the management of chronic obstructive pulmonary

disease.⁴ The implication is that spirometry testing is useful only when the patient has (or is suspected of having) established lung damage. Our results suggest that both these guidelines should be reviewed and that lung age testing (which is a quick, office based test that can be undertaken by a healthcare assistant) should be considered as part of a brief intervention package—either in all smokers over 35 (the lower age limit for this study) or all smokers. Currently the new contract for general practitioners in the UK includes incentives to confirm the diagnosis of chronic obstructive pulmonary disease with spirometry and to record smoking status in those with a record of relevant comorbidity (coronary heart disease, hypertension, diabetes, stroke, and asthma) and to give cessation advice. There is no incentive, however, to actively find cases of chronic obstructive pulmonary disease among smokers (or ex-smokers) in these high risk groups or in the general population. We recommend that the new UK NHS general practitioner contract should include incentives for spirometric assessment accompanied by individualised communication of lung age in smokers.

Our cost estimates, which assume that spirometry is carried out in UK general practice, suggest that estimation and communication of lung age is of comparable effectiveness to, and potentially cheaper than, other currently available treatments on the NHS, including nicotine replacement therapy,¹⁹ bupropion,²⁰ face to face counselling,²¹ and telephone counselling.²² Given the heavy health and economic burden of smoking, we believe that formal economic evaluation of this new and simple intervention should be a research priority.

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- 1 Van Schayck CP, Loozen JM, Wagena E, Akkermans RP, Wesseling GJ. Detecting patients at a high risk of developing chronic obstructive pulmonary disease in general practice: cross sectional case finding study. *BMJ* 2002;324:1370.
- 2 Price D, Duerden M. Chronic obstructive pulmonary disease. *BMJ* 2003;326:1046-7.
- 3 Soriano JB, Maier WC, Egger P, Visick G, Thakrar B, Sykes J, et al. Recent trends in physician diagnosed COPD in women and men in the UK. *Thorax* 2000;55:789-94.
- 4 National Collaborating Centre for Chronic Conditions. Chronic obstructive pulmonary disease. National clinical guideline on management of chronic obstructive pulmonary disease in adults in primary and secondary care. *Thorax* 2004;59(suppl 1):1-232.

- 5 Morris JF, Temple W. Spirometric "lung age" estimation for motivating smoking cessation. *Prev Med* 1985;14:655-62.
- 6 Bize R, Bumand B, Mueller Y, Cornuz J. Biomedical risk assessment as an aid for smoking cessation. *Cochrane Database Syst Rev* 2005;(4):CD004705.
- 7 Bednarek M, Gorecka D, Wielgomas J, Czajkowska-Malinowska M, Regula J, Mieszko-Filipczyk G, et al. Smokers with airway obstruction are more likely to quit smoking. *Thorax* 2006;61:869-73.
- 8 British Thoracic Society. Clinical information. COPD. www.brit-thoracic.org.uk/ClinicalInformation/tabid/66/Default.aspx.
- 9 Jones PW, Quirk FH, Baveystock CM. The St George's respiratory questionnaire. *Respir Med* 1991;85(suppl B):25-31.
- 10 Jones PW, Quirk FH, Baveystock CM. Why quality of life measures should be used in the treatment of patients with respiratory illness. *Monaldi Arch Chest Dis* 1994;49:79-82.
- 11 Fletcher C, Peto R. The natural history of chronic airflow obstruction. *BMJ* 1977;i:1645-8.
- 12 McClure JB. Are biomarkers a useful aid in smoking cessation? A review and analysis of the literature. *Behav Med* 2001;27:37-47.
- 13 Lancaster T, Stead LF. Physician advice for smoking cessation. *Cochrane Database Syst Rev* 2004;(4):CD000165.
- 14 Prochaska JO, DiClemente CC. Stages and processes of self-change of smoking: toward an integrative model of change. *J Consult Clin Psychol* 1983;51:390-5.
- 15 West R. *Theory of addiction*. 1st ed. Oxford: Blackwell, 2006.
- 16 West R, West R. Time for a change: putting the transtheoretical (stages of change) model to rest. *Addiction* 2005;100:1036-9.
- 17 National Institute for Health and Clinical Excellence. *Smoking cessation: draft guidance*. NICE, 2003. <http://guidance.nice.org.uk/page.aspx?o=429278>.
- 18 National Institute for Health and Clinical Excellence. *Brief interventions and referral for smoking cessation in primary care and other settings*. NICE, 2006. www.nice.org.uk/PHI001.
- 19 Silagy C, Lancaster T, Stead L, Mant D, Fowler G. Nicotine replacement therapy for smoking cessation. *Cochrane Database Syst Rev* 2004;(3):CD000146.
- 20 Hughes JR, Stead LF, Lancaster T. Antidepressants for smoking cessation. *Cochrane Database Syst Rev* 2004;(4):CD000031.
- 21 Lancaster T, Stead LF. Individual behavioural counselling for smoking cessation. *Cochrane Database Syst Rev* 2005;(2):CD001292.
- 22 Stead LF, Lancaster T, Perera R. Telephone counselling for smoking cessation. *Cochrane Database Syst Rev* 2003;(1):CD002850.

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11.8 Appendix 8. BMJ Editorial

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EDITORIALS

Incentives to quit smoking in primary care

Spirometry with pictorial feedback on lung age, not just raw data, improves quit rates



RESEARCH, p 598

Raphaël Bize senior research fellow

Jacques Cornuz associate professor, Department of Ambulatory Care and Community Medicine, University of Lausanne, Switzerland

raphael.bize@hospvd.ch

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In the accompanying randomised controlled trial, Parkes and colleagues assess the effect of telling patients over 35 years of age their estimated spirometric lung age as an incentive to quit smoking.¹ Support for conducting the trial comes from a recent Polish observational study on the potential association between smoking cessation and participants' spirometry results, as communicated using Fletcher and Peto's diagram (a pictorial representation of how smoking ages the lungs).² The Polish study showed higher smoking cessation rates at one year in smokers with airway obstruction than in those with normal spirometric parameters. However, the study had no control group without spirometry testing or without feedback on such testing. The authors called for a large randomised controlled trial comparing the effect of providing spirometry results versus no spirometry results on smoking cessation.

In Parkes and colleagues' trial, participants in the intervention group received comprehensive information about their spirometry results including individualised interpretation, estimated lung age, and Fletcher and Peto's diagram. People in the control group received written results as raw data on forced expiratory volume in one second, with no further explanation. Participants in both groups were advised to quit smoking and were offered an optional referral to an intensive support service. Smokers randomised to the intervention group were about twice as likely to be not smoking at 12 months' follow-up than those in the control group. A subgroup analysis found no evidence of a dose-response relation between "lung age deficit" (lung age minus chronological age) and the outcome, as quitters and non-quitters had similar lung age deficits. This study did not look at the potential health benefit of screening for chronic obstructive pulmonary disease because all participants underwent spirometry testing.

Another recent randomised controlled trial investigated a closely related research question in smokers aged 18-24 years.³ It focused on intermediate psychological outcomes, based on health behaviour theories such as the "health belief model." This model states that people are likely to follow a particular health action if they think they are susceptible to a condition that they consider serious, and if they believe that the benefits of the action outweigh the costs.⁴ The intervention group received a smoking cessation booklet plus feedback about their spirometric lung age and respiratory symptoms, and the control group received only the

smoking cessation booklet. Perceived risks, worries, and desire to quit smoking were assessed using 7 point Likert-type scales at study entry and after delivery of the intervention. No significant differences were found between groups at either time point. They also assessed the perceived relevance of lung age and feedback on respiratory symptoms in the intervention group using the "10 item personal involvement inventory." A significant inverse correlation was seen between lung age deficit and perceived relevance of lung age feedback, perhaps as a defensive reaction against potentially worrying information.

A systematic review explored the effect of biomedical risk assessment as an aid for smoking cessation.⁵ It included trials in which a measurement—such as exhaled carbon monoxide, spirometry, or genetic testing—was used to increase motivation to quit. For trials to be eligible, the control group had to receive all parts of the intervention except for the biomedical feedback. Only one trial of spirometry was eligible for this review.⁶ It found no significant difference in smoking cessation at 12 months' follow-up between participants receiving spirometry feedback and repeated counselling and those receiving counselling but not spirometry testing (odds ratio for 7 day abstinence at 12 months in the intervention group compared with the control group 1.21, 95% confidence interval 0.60 to 2.42). An ongoing updated search found another eligible paper that had similar results.⁷ Parkes and colleagues investigated a slightly different research question (comprehensive, illustrated, and individualised oral feedback versus short, raw, and written feedback) than these two trials where participants did not undergo spirometry if they were allocated to the control condition.

On the basis of the evidence so far, general practitioners have to decide whether to wait for a trial comparing the potential benefit for smoking cessation of spirometry testing using lung age feedback versus no spirometry testing or whether to adopt the strategy suggested by Parkes and colleagues. In making this decision they should be aware of the limitations of the trial—for example, the lack of information about the comparability of the study sample with the entire recruitment population, the longer duration of contact between participants and caregivers in the intervention group than in the control group, and outcome data that are limited to point-prevalence abstinence.⁸ Despite these limitations, however, providing feedback on lung age with graphic displays seems to be the best option

so far for communicating the results of spirometry. This strategy might also be an opportunity for general practitioners to tailor smoking cessation messages to the individual, as recommended in the recent National Institute for Health and Clinical Excellence (NICE) guidance on smoking cessation.⁹

- 1 Parkes G, Greenhalgh T, Griffin M, Dent R. Effect of telling patients their lung age on smoking quit rate: randomised controlled trial. *BMJ* 2008 doi: 10.1136/bmj.39503.582396.25.
- 2 Bednarek M, Gorecka D, Wielgomas J, Czajkowska-Malinowska M, Regula J, Mieszko-Filipczyk G, et al. Smokers with airway obstruction are more likely to quit smoking. *Thorax* 2006;61:869-73.
- 3 Lipkus IM, Prokhorov AV. The effects of providing lung age and respiratory symptoms feedback on community college smokers' perceived smoking-related health risks, worries and desire to quit. *Addictive Behav* 2007;32:516-32.
- 4 Conner M, Norman P. *Predicting health behaviour*. 2nd ed. Maidenhead: Open University Press, 2005.
- 5 Bize R, Burnand B, Mueller Y, Cornuz J. Biomedical risk assessment as an aid for smoking cessation. *Cochrane Database Syst Rev* 2005;(4):CD004705.
- 6 Segnan N, Pontl A, Battista RN, Senore C, Rosso S, Shapiro SH, et al. A randomized trial of smoking cessation interventions in general practice in Italy. *Cancer Causes Control* 1991;2:239-46.
- 7 Buffels J, Degryse J, Decramer M, Heyman J. Spirometry and smoking cessation advice in general practice: a randomised clinical trial. *Respir Med* 2006;100:2012-7.
- 8 West R, Hajek P, Stead L, Stapleton J. Outcome criteria in smoking cessation trials: proposal for a common standard. *Addiction* 2005;100:299-303.
- 9 National Institute for Health and Clinical Excellence. *Public health guidance 10. Smoking cessation services in primary care, pharmacies, local authorities and workplaces, particularly for manual working groups, pregnant women and hard to reach communities*. 2008. www.nice.org.uk/PH010.

11.9 Appendix 9. Rapid responses

Usefulness of lung age

7 March 2008

Hiroshi Kawane,
Professor
The Japanese
Red Cross
Hiroshima
College of
Nursing,
Hatsukaichi City,
Hiroshima, 738-
0052, Japan
Send
response to
journal:
Re:
Usefulness of
lung age

The research by Parkes et al[1] may suggest that people tend to care about their age and want to be looking younger. Morris et al[2] reported the usefulness of lung age for motivating smoking cessation. We also made a calculation formula of lung age for the Japanese and found it useful for smokers with mild airflow obstruction[3]. If the lung age was calculated for patients with chronic obstructive pulmonary disease, it may be estimated at 100 or over. Thus in some cases, using lung age for smoking cessation advice proved to be impractical.

References

- [1]Parkes G, Greenhalgh T, Griffin M, Dent R. Effect on smoking quit ratio of telling patients their lung age: the Step2quit randomised controlled trial. *BMJ* 2008;0:bmj.39503.582396.25v1
- [2]Morris JF, Temple W. Spirometric "lung age" estimation for motivating smoking cessation. *Prev Med* 1985;14:655
- [3]Kawane H, Kojima K. Calculation of "lung age" from lung function. *Rinshokensa* 1998;42:248

Competing interests: None declared

comparison with counselling

8 March 2008

Rajasree Pal R,
Lecturer
Dr SMCSIMCh
Send
response to
journal:
Re:
comparison
with
counselling

The study does not compare the effect of patient counselling with showing images of lung age. The paper compares FeV1 with lung age which points to the fact that patients tend to understand simpler terms and images than complicated parameters like FeV1.

Competing interests: None declared

Age to peak flow readings?

9 March 2008

John J.
Kivlighan,
Retired
Marlborough,
MA 01742
Send
response to
journal:
Re: Age to
peak flow
readings?

Is there a chart/scale of age to peak flow readings that a layman could use? Many ex-smokers and smokers with asthma have personal peak flow meters and such a chart could be very useful to laymen at large.

Regards, John K.

Competing interests: None declared

"lung age" is both physical and mental index

9 March 2008

Xiaofeng Ren,
Associate
Professor
Microbiology
Lab, Northeast
Agricultural
University, 59
Mucai Street,
150030 Harbin,
China
Send
response to
journal:
Re: "lung age"
is both
physical and
mental index

The work of Parkes et al. proves the positive effect of telling smokers their lung age significantly improves the likelihood of them quitting smoking[1], however, their report just highlights a phenomenon. The question "why" is not clear currently.

In my opinion, the "lung age" do display the physical condition of the smokers and it is detectable by clinical examination and can be used as evidences for related evaluation and therapy. Nevertheless, once the smokers were informed on the status of their "lung age", it would be a very important mental stress that is usually difficult to be found and will lead to different response to any advices and intervention. The expected responses may be dependent on the good or bad status of smokers' lung age. Correspondingly, under most circumstances, the smokers may give rise to a very positive response, if they know that they have poor "lung age".

Although "lung age" should be both physical and mental index for patients including long-history smokers, some of the patients usually do not know the exact status of their "lung age". Particularly, some of the patients do not wish to express the weakness of their mentalness frankly. In other words, some of them may do not accept/believe the doctors' advices. Therefore, cooperation between the clinician and psychologists is needed for helping the patients in therapy and also for elucidating the hypothesis furtherly.

Reference

[1] Parkes G et al., Effect on smoking quit rate of telling patients their lung age: the Step2quit randomised controlled trial. BMJ, doi:10.1136/bmj.39503.582396.25 (published 6 March 2008).

Competing interests: None declared

Re: Age to peak flow readings?

10 March 2008

Gary Parkes,
GP, Researcher
EN118EP

Send
response to
journal:

Re: Re: Age to
peak flow
readings?

Some attempts have been made by other researchers to correlate FEV1 with PEFR but this has never become an established method. Therefore there is no reliable formula to make the conversion from PEFR into lung age. There are well-established diagnostic criteria for diagnosing COPD using spirometry measurements (FEV1 and FEV1/FVC ratio) but PEFR cannot be used for this purpose. PEFR is very valuable for diagnosis and monitoring of asthma.¹

Lung age estimation was developed from reference linear regression equation tables and found to be correlated best with FEV1.²(Morris and Temple)

Reference List

- 1.Diagnosing COPD. Thorax 59.90001 (2004): i27-i38.
- 2.Morris, J. F. and W. Temple. "Spirometric "lung age" estimation for motivating smoking cessation." Preventive Medicine 14 (1985): 655-62.

Competing interests: Author of the article

Lung age by spirometry

17 March 2008

Anthony N
Glaser,
Assistant
Professor of
Family Medicine
Medical
University of
South Carolina,
SC 29483

Send
response to
journal:

Re: Lung age
by spirometry

Parkes et al's paper is encouraging. I have been doing routine spirometry on smokers for some time, and showing patients the Fletcher curve, and anecdotally it seems to have some impact. But it would be helpful to be able to tell patients their lung age. The formulae are cumbersome and the statement "In practice, the lung age is automatically generated by adjustment of the settings of the spirometer" is not very revealing. Just how can lung age be automatically generated by the spirometer? Orr can someone please write a small Palm program that calculates lung age with the stated formulae?

Competing interests: None declared

Re: Lung age by spirometry-reply-re:calculator

17 March 2008

Gary Parkes,
GP &
Researcher
Limes Surgery,
Hoddesdon

Send
response to
journal:

Re: Re: Lung
age by
spirometry-
reply-
re:calculator

The Step2quit project used modern spirometers capable of displaying, downloading and/or printing graphs and has settings that can be adjusted to convert FEV1(knowing height and gender) into lung age.However, older models and hand held spirometers do not have that option.

I agree that the mathematical formula (as per Morris and Temple) is not practical for daily use in a busy clinic. However, I can supply a calculator (in microsoft excel), which you should be able to keep on your computer desktop or palmcomputer. You only have to type-in the height and FEV1 to get the result.

Just e mail me on the address given in the article and I will send you a copy.

Dr Manoj K.
Saraswat,
SpR
General/Geriatric
Medicine
Scarborough
Gen.Hospital
YO12 5DX,
Dr Shweta
Saraswat, ST1
Psychiatry:Royal
Edinburgh
Hospital
Send
response to
journal:
[Re: Looking
Beyond Lung
Age](#)

Dear Sir,

We read the results and methodology of Step2quit randomised controlled trial by Parkes et al with great interest. The study results are encouraging as significantly more participants quit smoking in the intervention arm, which were explained their lung age based on spirometric results of their lung function tests.

Smoking cessation advice is something that almost all clinicians routinely deliver across the specialities and to date most of the guidance suggested to use combination of Intensive behavioural support and nicotine replacement treatment to achieve highest quit rate(1) Although the results from Step2quit randomised trial are encouraging several questions remain to be answered:

1. The study included a very select population from a distinct geographic area of UK.
2. Patients with mental health problems were not included in the study, this group has particularly high prevalence(2)
3. Outcome data are limited to point prevalence on abstinence and relapse rate after Quitting smoking are quite high(3)
4. We are not sure how applicable is to calculate lung age from the seemingly complex formulae given in the article

We think more research is needed to clearly validate the usefulness of one strategy over the other in different population groups and patients with different background Social- Medical conditions

References: 1.Raw et al Thorax 1998;53(suppl 5,part1)S1-19

2.Karen Lasser et al, JAMA 2000;284:2606-2610

3.Results from NHANES Population Survey:The health benefits of smoking cessation:Washington DC,DHHS,1990

Competing interests: None declared

Jay R. Schrand,
Systems
Engineer,
Independent
Researcher
Enid, OK 73701

Send

response to
journal:

Re: Stress,
Tobacco
Control and
the Insula

Parkes⁽¹⁾ et al tested the use of a psychological persuasion technique on those who smoke. Revealing a hypothetical age of the lungs increased the percentage of quitters from 6.4% to 13.6%. Stress is indeed a motivator. A strong dose of guilt with claims of better health does sell medicine and procedures. However as with other cessation methods, a failure rate of 86.4%, is still abysmal, and reflects a lack of understanding of the complete relationship between tobacco use and the respiratory system.

Stress, Respiration and Tobacco Use (see [Figure 1](#))

Traumatic stress in the presence of chronic stress induces asthma attacks⁽²⁾. Felitti et al found a strong relationship between the stress of Adverse Childhood Experiences (ACE's) and several of the leading causes of death in adults such as stroke (ACE's >4, OR= 2.4) and Chronic Obstructive Pulmonary Disease(COPD) (ACE's >4, OR= 3.9)⁽³⁾. These exposed to the stress of ACE's, have a strong graded relationship with eventual tobacco initiation⁽⁴⁾.

Tobacco has been used for 2000 years by the Native American population to treat respiratory disorders⁽⁵⁾ and by physicians in the late 1800's to treat asthma⁽⁶⁾. Nicotine is a CNS and respiratory stimulant, increasing respiratory drive⁽⁷⁾. Differences in respiratory symptoms appear in childhood before tobacco initiation⁽⁸⁾. However, Tobacco Control (TC) studies appear to ignore the likelihood that many of those who smoke tobacco are unknowingly self-medicating stress induced respiratory disorders.

Asthma, COPD and Smoking

Data from a recent study⁽⁹⁾ by Silva et al. indicates that when asthma is included in the

Data from a recent study⁽⁹⁾ by Silva et al. indicates that when asthma is included in the model, the risk factor for smoking on COPD drops from the usual 5-13 range to a still significant but meager 2.9 compared with 12.5 for lifetime asthma. Though not discussed in the text, the newly discovered confounding relationship suggests that asthma is a greater risk factor for COPD rather than smoking. This is consistent with nicotine's positive influence on respiration.

The Insula and Tobacco Control Methods

The insula translates sensory perceptions (smell, sight, etc) into feelings [love << like << ambivalence >> disgust >> hate] that motivate human choice. A stroke in the insular area often results in a shift of this reference point. What was once pleasurable now elicits no feelings or even disgust. Naqvi et al discovered that those with an insular stroke find it easy to quit⁽¹⁰⁾.

While recognizing that brain surgery to produce an insular stroke for the purpose of cessation would be unethical, the authors speculate that further experiments are warranted in the "human laboratory".

In the Parkes et al study⁽¹⁾, the high number with CVA and stroke in the control group at baseline with an increased likelihood of cessation would tend to decrease the apparent program effect. It would likely be an artifact.

However, the TC program depends heavily on social force and psychological guilt. This negative reinforcement combined with the low likelihood of successful cessation induces stressful conflict. This would be especially felt by the most vulnerable with a high number of ACE's and have difficulty with the program. Stress induction, so similar to the methods used in dysfunctional family environments, increases the risk for stress

related illnesses including stroke, and may actually be the basis for many of the current cessations.

This analysis indicates that insular stroke likely plays a part in the natural and program tobacco cessation process. The only question is: "How much?" Any experimental techniques in the human laboratory must test for potential iatrogenic effects. Follow-up needs to occur to determine how many of the cessation successes are because of (silent) insular stroke.

REFERENCES

1. Parkes G, Greenhalgh T, Griffin M, Dent R.

Effect on smoking quit rate of telling patients their lung age: the Step2quit randomized controlled trial.

BMJ, doi:10.1136/bmj.39503.582396.25

2. Sandberg S, Paton JY, Ahola S, et al.

The role of acute and chronic stress in asthma attacks in children.

Lancet. 2000 Sep 16;356(9234):982-7.

3. Felitti VJ, Anda RF, Nordenberg D, Williamson DF, Spitz AM, Edwards V, Koss MP, Marks JS.

Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. The Adverse Childhood Experiences (ACE) Study.

Am J Prev Med. 1998 May;14(4):245-58.

4. Anda RF, Croft JB, Felitti VJ, Nordenberg D, Giles WH, Williamson DF, Giovino GA.

Adverse childhood experiences and smoking during adolescence and adulthood.

JAMA. 1999 Nov 3;282(17):1652-8.

5. Gandevia B.

Historical review of the use of parasympatholytic agents in the treatment of respiratory disorders.

Postgrad Med J. 1975;51(7 SUPPL):13-20.

6. National Library of Medicine - Elizabeth Fee, Ph.D. Exhibition Director

Breath of Life - Asthma: From Symptom to Disease - Smoking Your Asthma Away

http://www.nlm.nih.gov/hmd/breath/breath_exhibit/Asthma/smoking/smokingframe.html
(accessed 03/13/08)

7. Haxiu M A, van Lunteren E, Van de Graff W B, Strohl K P.

Action of nicotine on the respiratory activity of the diaphragm and genioglossus muscles and the nerves that innervate them.

Respir Physiol 1984; 57: 153

8. Lebowitz MD.

Smoking habits and changes in smoking habits as they relate to chronic conditions and respiratory symptoms.

Am J Epidemiol. 1977 Jun;105(6):534-43.

9. Silva GE, Sherrill DL, Guerra S, et al.

Asthma as a Risk Factor for COPD in a Longitudinal Study.

Chest. 2004 Jul;126(1):59-65.

10. Naqvi NH, Rudrauf D, Damasio H, Bechara A.

Damage to the insula disrupts addiction to cigarette smoking.

Science. 2007 Jan 26;315(5811):531-4.

Figure 1 - Graphic

Competing interests: In 1994, the author requested an educational grant from the Council for Tobacco

Research to study the relationship between tobacco use and sleep apnea. This request was denied.

However the letter seems to have become part of the tobacco archives:

<http://tobaccodocuments.org/ctr/60024574-4574.html> Aside from this, the author has no financial interest

in the tobacco, diet, or health industries other than as a consumer of their products and services.

Lung age by spirometry

28 March 2008

Kathy Hartley,
Specialist
Trainee in Public
Health
Suffolk PCT,
Rushbrook
House,
Papermill Lane,
Bramford,
Ipswich, IP8
4DE,
Daniel G
Showell, and
Padmanabhan
Badrinath
Send
response to
journal:
[Re: Lung age
by spirometry](#)

We have some methodological reservation about the recent and important randomised controlled trial, Step2quit[1]. There are two threats to the internal validity of this study. Firstly, the additional verbal interaction (page 599) given to the intervention group as compared to minimal interaction given to the control (page 599) may have biased the outcome. It is plausible that this difference in the way participants received the intervention could have changed their behaviour to some extent.

Secondly, the intervention was not the single intervention of telling smokers their lung age. It consisted of giving graphical as well as personalised information about lung age. Either or both of these may be responsible for the outcome. Indeed the Fletcher and Peto graph[2] is used in group smoking cessation classes without personalised data.

It was helpful to have the numbers needed to treat (NNT) of 14 though the 95% confidence intervals for this would also have been of interest. We make these to be 8 to 51.

We feel that it is important that there is more clarity on the first two issues above before this intervention is adopted widely in practice.

[1] Parkes G, Greenhalgh T, Griffin M, Dent R. Effect on smoking quit ratio of telling patients their uc age: the stpe2quit randomised controlled trial. 2008;336:598-600 [2] Fletcher C, Peto R. The natural history of chronic airflow obstruction. BMJ 1977;i:1645-8.

Competing interests: None declared

Gary Parkes,
GP, Researcher
EN118EP,
Trisha
Greenhalgh,
Mark Griffin
Send
response to
journal:
Re: Re: Lung
age by
spirometry

Dr Kathy Hartley et al are correct in their statement that the intervention in the step2quit study is more than just giving lung age measurements.(1) However, it is not correct to say that this is methodological bias. Bias is defined as 'any systematic error in the design, conduct or analysis of a study that results in a mistaken estimate of an exposure's effect'.(2) Although the abstract summarizes the intervention as 'giving results in terms of lung age', it is always important to look beyond the abstract to fully understand the methodology of a piece of research.

I hope Dr Hartley et al will agree that the full text version of the research intervention clearly and openly states that: 'Participants in the intervention group were given their results verbally, immediately after randomisation, in the form of "lung age" with a graphic display'. And 'Written results were given to the intervention group as lung age'. This is not bias, this IS the (complex) intervention. Anyone wanting to introduce the intervention in their own clinical practice must be careful not to shortcut the intervention to just a verbal delivery of 'lung age'.

The complex intervention that produced the dramatic effect on quit rate in this study has three important components: verbal information, graphic information, and an individualised results letter. Together, these interventions seem to help people think about their health in a personalised way. We are continuing to explore the psychological literature to develop a theory of change as to why this may be.

Clinicians will need to consider the 'whole package' if they wish to get similar favourable outcomes. To this end, I have requested the assistance of Health Enterprise East (<http://www.hee.org.uk/>) to create a clinical pack to make it easy to deliver the triple intervention on a wider scale.

Based on our 95% CI for the ARR (2.2% to 12.1%) the 95% CI for the NNT comes out to be 9 to 46.

Gary Parkes, Trisha Greenhalgh and Mark Griffin

Even if you do not have a spirometer which can be set to display lung age the calculation derived from Morris and Temple(3) can be done easily using the excel spreadsheet now downloadable for free at www.step2quit.co.uk, and the graphics can be downloaded as Powerpoint slides from BMJ.com.

Reference List

(1) Parkes G, Greenhalgh T, Griffin M, Dent R. Effect on smoking quit rate of telling patients their lung age: the Step2quit randomised controlled trial. *BMJ* 2008; 336(7644):598-600.

(2) Gordis L. Bias, Confounding, and Interaction. *Epidemiology*. Second ed. Philadelphia: W.B.Saunders Company; 2000.

(3) Morris JF, Temple W. Spirometric "lung age" estimation for motivating smoking cessation. *Preventive Medicine* 1985; 14:655-662.

Competing interests: Authors of the original research

Why the new UK NHS GP contract should not include incentives for spirometric assessment in smokers

8 April
2008

Alberto Donzelli,
Director of
Health
Education
Service - ASL
Città di Milano -
Italy
via Ricordi 1 -
20131 Milano -
Italy
Send
response to
journal:
Re: Why the
new UK NHS
GP contract
should not
include
incentives for
spirometric
assessment in
smokers

A Cochrane Systematic Review (1) concluded "Current evidence of lower quality does not ... support the hypothesis that biomedical risk assessment increases smoking cessation in comparison with standard treatment".

Exhaled CO measurement and spirometry were used together in three trials, no one resulting in statistically significant differences between intervention and control groups. Two out of three trials even show a trend towards a worsening in the ORs (95% CI) of smoke quitting: 2.45 (0.73 to 8.25) and 3.50 (0.88 to 13.92) with CO measurement plus spirometry. Another RCT with only spirometry has an OR 1.21 (0.60-2.42) (and another with ultrasonography of carotid and femoral arteries performed in light smokers, smoking on average 10 to 12 cigarettes a day, significantly worsened the OR of smoke quitting: 3.15 (1.06 to 9.31), showing the actual risk to raise costs and harms at the same time (1).

Parkes et al (2) state that NICE guidelines (which do not mention spirometry testing at all) should be reviewed and that "lung age" testing should be considered a part of a brief intervention package either in all smokers over 35 or all smokers. And they recommend that the new UK NHS general practitioner contract should include incentives for spirometric assessment accompanied by individualised communication of lung age in smokers.

Conclusions (and claim) expressed in such way seem very little supported by trial results. Indeed Parkes et al performed a spirometric assessment to all participants and the control group was the communication: an information given immediately, in the form of "lung age" with a graphic display, followed by a written personalised letter in the intervention group; a letter delayed by several weeks with results given as simple FEV1 with no further explanation in the control group.

The trial does not support neither the belief that the information of a worse spirometric lung age raises the patient chances of quitting smoking. On the contrary, the opposite seems true as trend (the mean lung age deficit was 8.7 years and 9.4 years in the quitters and non-quitters).

Thus, what are the data telling us?

1) How the authors acknowledge, the success was likely to depend on communication: how the information was conveyed and understood, also using graphic displays and individualised letters. Yet, is questionable if a spirometric assessment is needed, instead of less expensive tools.

n.b.: a more effective communication has perhaps also caused, as trend, a wider use of effective clinical and pharmacological support to quit: 7.8% in control group and 10.7% in the intervention group. Anyhow, quit rates at 12 months in the intervention group (13.6%) are similar to those obtained with effective available treatments, and less than the results achieved in a trial performed in the primary care setting (3)).

2) On the basis of existing evidence it is hard to recommend a spirometric assessment (and with which interval?) for all smokers over 35 (or all smokers at all). A spirometer is a technological device involving various costs: price (about € 1000-2000 in the Italian market), necessity of training to use it correctly, reimbursement fees of € 51,65 by Italian NHS or fees paid out of pocket by patients, and a performing time (30 minutes

for every spirometry) which goes unavoidably in competition with other services of proven effectiveness. If the GP wants support for his/her quitting advice and counselling, with a graphic display, this could be the illustration/delivery of risk charts (in Italy there are specific and impressive colourful charts elaborated by Superior Institute of Health, by age, sex and smoking status for COPD, for lung cancer and for global cardiovascular risk, usable also in combination). It is questionable if we need a spirometry to convey an effective message on the almost sure effects (either pulmonary, or cardiovascular, or tumoral, or overall) for smokers who do not want to quit: a more direct, simple, multimedial educational intervention may be a real research priority.

For GPs who want a technological support and to follow the evidence of Parkes' trial, can be enough a simple FEV1 meter (about € 50 in the Italian market), which gives the essential information needed to show in colourful graphs the "lung age" (n.b.: the high reliability of measure is not the determinant factor).

Finally, in my opinion the incentives (in proper amounts) should be better deserved by GPs whose formally reported interventions can document the real outcome of interest, that is a long term quitting status in every patient who was a previously certified smoker. Instead, incentives should not be provided for services or processes which per se are not a guarantee of outcome, and that can lead to technological abuse and interest-driven behaviours of providers.

Dr. Alberto Donzelli
Director of Health Education Service
ASL Città di Milano
adonzelli@asl.milano.it

(1) Bize R et al. Biomedical risk assessment a san aid for smoking cessation.
Cochrane Database of Systematic Reviews 2005, issue 4. Art. N°: CD004705

(2) Parkes G et al. Effect on smoking quit rate of telling patients their lung age: the Step2quit randomised controlled trial. BMJ 2008; 336: 598- 600

(3) Fossati R et al. A double-blind, placebo controlled, randomized trial of bupropion for smoking cessation in primary care. Arch Intern Med 2007; 167:1791-7

Competing interests: None declared

Philip H Quanjer,
retired
7443 GT
Nijverdal, the
Netherlands
Send
response to
journal:

Re: Should we
use lung age?

I read with interest Parkes' study [1] about lung age and fully sympathise with efforts to make people give up smoking. However, should doctors use biased information to persuade smokers? Example: a man, height 178 cm, age 50 year, FEV1 3.20 litre. Predicted FEV1 according to ECCS/ERS [2], Crapo [3] and Morris [4] comes to 3.71, 3.94 and 3.59 litre, respectively. The observed FEV1 is well within the normal range for healthy lifelong nonsmokers. "Lung age" according to the above 3 reference equations is 68, 83 and 62 year, respectively; so, what is this person's "lung age"? If the person does not smoke we regard the FEV1 as being within the normal range and unremarkable. If the subject smokes, we no longer attribute the difference between measured and predicted value to normal variation but suggest that it is all accounted for by smoking. We introduce further bias by only attributing a difference from predicted to smoking if the recorded FEV1 is less than predicted. We inform only smokers about a fictitious lung age and withhold information about the normal range. Does the end really justify the means, is this a desirable role model for the doctor-patient relation?

1. Parkes G, Greenhalgh T, Griffin M, Dent R. Effect on smoking quit rate telling patients their lung age: the Step2quit randomized controlled trial. *BMJ*. 2008;336:598-600.

2. Quanjer PhH, Tammeling GJ, Cotes JE, Pedersen OF, Peslin R, Yernault JC. Lung volume and forced ventilatory flows. Report Working Party Standardization of lung function tests; Official Statement European Respiratory Society. *Eur Respir J* 1993; 6 Suppl 16:15-40.

volume and forced ventilatory flows. Report Working Party Standardization of lung function tests; Official Statement European Respiratory Society. *Eur Respir J* 1993; 6 Suppl 16:15-40.

3. Crapo RO, Morris AH, Gardner RM. Reference spirometric values using techniques and equipment that meet ATS recommendations. *Am Rev Respir Dis* 1981;123:659-664.

4. Morris JF, Temple WP, Koski A. Normal values for the ratio of one- second forced expiratory volume to forced vital capacity. *Am Rev Resp Dis* 1973;108:1000-1003.

Philip H. Quanjer

Competing interests: None declared

Study's conclusion about screening is unwarranted

16 April 2008

Kenneth W Lin,
Medical Officer
Agency for
Healthcare
Research and
Quality,
Rockville,
Maryland USA
20850

Send
response to
journal:
[Re: Study's
conclusion
about
screening is
unwarranted](#)

Although this is an interesting study, the results only support the conclusion that for asymptomatic smokers who undergo spirometry, communication about lung age is a more effective motivator for tobacco cessation than uninterpreted spirometry measurements. It does not establish the independent motivational effectiveness of doing spirometry screening vs. not doing it, which would require a randomized trial in which the control arm did not receive spirometry at all. Only 2 randomized controlled trials of this type have been conducted and both had negative results. For this and other reasons, the U.S. Preventive Services Task Force recently recommended against screening adults (regardless of smoking status) for COPD using spirometry (1).

1. U.S. Preventive Services Task Force. Screening for chronic obstructive pulmonary disease using spirometry: U.S. Preventive Services Task Force recommendation statement. *Ann Intern Med* 2008;148:529-34.

Competing interests: None declared

Lung age should be compared to FEV1 percent predicted

5 May 2008

Rei Suganaga,
senior resident
Kameda Medical
Center,
Kamogawa
Chiba 296-8602
Japan,
Makito Yaegashi

Send
response to
journal:
[Re: Lung age
should be
compared to
FEV1 percent
predicted](#)

Parks et al have shown the usefulness of lung age in smoking cessation. However, is there an easier way to tell patients understandable numbers?

There are 9 randomized-controlled trials addressing whether the use of spirometry will contribute to smoking cessation [1]. However, some study did not state how the result conveyed to patients [2], while other conveyed only whether patients had obstruction or not [3]. Since this is a matter of education, how the results conveyed to the patients should be clearly stated.

FEV1 presented as percent predicted (i.e. 70% predicted) for their age, height, sex and race are always included in the spirometry reports. Knowledgeable physicians are using percentage rather than raw numbers while they are explaining the spirometry results. Instead of comparing lung age to FEV1 percent predicted, Parks et al used FEV1 presented as a raw number (i.e. 1.5L). Lay people would not understand the meaning and normal value of FEV1. The significant difference presented in this study could be solely from the use of a number that has little meaning to most patients.

Lung age should therefore be compared to FEV1 percent predicted to elucidate the more effective way to convey understandable information to patients.

1) Kenneth Lin et al. Screening for Chronic Obstructive Pulmonary Disease Using Spirometry: Summary of the Evidence for the U.S. Preventive Services Task Force. *Ann Intern Med*. 2008; 148 : 535-543

2) Johan Buffels et al. Spirometry and smoking cessation advice in general practice: A of randomized clinical trial. *Respiratory Medicine*. 2006; 100: 2012-017

3) Jeffrey M. Sippel et al. Smoking Cessation in Primary Care Clinics. *J Gen Intern Med*. 1999; 14: 670-676

Competing interests: None declared

11.10 Appendix 10. Other feedback

11.10.1 Centre for Evidence-Based Medicine, Oxford

Dear Dr Parkes

My GP practice journal club session today we looked at your recent trial in the BMJ on telling smokers their lung age. We were all enthused by the idea of integrating this into the overall practice, but had a few questions we wanted to ask.

1. Could you let us have an example of the letter you sent at 4 weeks? Was it simply the lung age plus:

‘This type of lung function test does not tell us anything about the risk of other serious diseases related to smoking such as lung cancer or heart disease or stroke. Smoking cessation is therefore still important for all people regardless of their age or the results of these lung tests.’ and the smoking cessation clinic number?

2. You sent the letter to all current smokers, but we wondered how to make this part on an ongoing practice routine? We considered just doing it when smokers presented (and perhaps repeating every 5 years) but wondered if you other ideas?

Also out of curiosity, can I ask if you have had others approach about details of the ‘how to’?

Congratulations on a really useful study,

Regards

Paul Glasziou

Director, Centre for Evidence-Based Medicine,

Department of Primary Health Care,

University of Oxford

11.10.2 Islington PCT

Subject: query re your research

Date: Tue, 10 Jun 2008 12:07:01 +0100

From: mary.price@islingtonpct.nhs.uk

To: parkesko@hotmail.co.uk

Dear Gary,

I read with interest you work re effect on quit rate of telling patients their lung age. Can you tell me how the cost per successful quitter of £280 is reached? I am interested to know what the capital and revenue costs in equipment and additional personnel are in order to make a business case for doing something similar.

Many thanks

Mary Price

Long Term Conditions and Access Coordinator

Islington PCT

11.10.3 Barnet PCT

Dear Gary

I was very interested in the recent BMJ article (14th April) – 'Effect on smoking quit rate of telling patients their lung age' and wish to prepare a business case for lung age testing/screening to be offered to all clients accessing our Stop Smoking Services. This will involve rolling out the scheme to a large number of GP practices, pharmacies and a small number of clinics.

I would be very grateful for your advice and would appreciate if you could provide the following information detailed below.

- Cost of Spirometer used and parameters you used in choosing the

Spirometer. Would you recommend it for our purposes?

- Whether you would advise one Spirometer per GP practice or one for each practitioner providing smoking cessation advice and treatment
- What is content/involved in the training, cost of training, how training was delivered e.g. number trained per session (we would need to train GP's practice nurses, pharmacists and smoking cessation clinic advisors) and duration of training sessions
- Estimated time of screening test and explanation of result to client - in the paper it states 30 minutes to perform the test and a further 15 minutes per patient reviewing results. Please can you give me some details of why it takes this length of time as this will be an issue for GP's, practice nurses and pharmacists in terms of capacity to incorporate this test into their current practice
- What information is given to the clients about the test?

Many thanks

Kind Regards

Susan Hearn
Lifestyle Services Manager
Health Improvement Directorate
Barnet Primary Care Trust
1st Floor, Deansbrook House
Edgware Community Hospital
London
HA8 0AD

11.10.4 ACP Journal Club

Dear Dr. Parkes

Below is the URL of the abstract and commentary for your recent research that was featured in ACP Journal Club. Thank you very much for your collaboration in the process of abstracting and commenting on your important work.

<http://www.acpjc.org/Content/149/1/issue/ACPJC-2008-149-1-005.htm><http://>
<http://>When opening your abstract you will be asked for a username and password. the username is ***** and the password is *****

Below is the link to the commentator form for completion.

<http://plus.mcmaster.ca/commentators/form/acp.aspx>

Yours sincerely,

R. Brian Haynes, MD, PhD

Editor

Evidence-Based Journals

Health Information Research Unit

McMaster University

1200 Main Street West, HSC-3H7

Hamilton, Ontario, CANADA L8N 3Z5

Phone: 905-525-9140 Ext 22737

Fax: 905-546-040

11.10.5 Evidence Based Nursing

Dear Dr. Parkes,
The Limes Surgery,

RE: Your article: Effect on smoking quit rate of telling patients their lung age:

the Step2quit randomised controlled trial. BMJ. 2008;336:598-600.

In addition to your captioned article being chosen for abstraction for ACP Journal Club, it was also chosen for Evidence-Based Nursing (<http://ebn.bmj.com/>). Evidence-Based Nursing is a quarterly publication of the BMJ Publishing Group. Its purpose is to help nurses keep up to date by abstracting high quality articles from key journals of relevance to nursing practice. Your article was chosen for abstraction because it met (at least) these criteria:

- i. random allocation of participants to comparison groups;
- ii. follow up (end point assessment) of at least 80% of those entering the investigation;

- iii. outcome measure of known or probable clinical importance;
- iv. analysis consistent with study design.

Attached is a structured abstract plus expert commentary of your study. If you have problems opening this file, please let us know right away and we will resend in another format.

The purpose of this letter is to give you the opportunity to please verify the accuracy of our version of your work before we go to press. In the abstract we may have included some calculations based on the data in your article. The redline feature is turned on so that you can make any corrections directly in the abstract. Please do not turn off the feature so that your edits will clearly be noted.

Our Glossary can be viewed at

http://hiru.mcmaster.ca/EBJournals/EBN_AE-Glossary.pdf

If you have any comments or corrections for the abstract that we have prepared or for the expert commentary, please contact Joanne Gunby. Joanne can be reached by telephone ((905) 525-9140 x22091), fax (905-546-0401), or e-mail (gunbyj@mcmaster.ca).

Because we have a deadline to meet, we will need to assume our version is acceptable unless we hear from you within two working days of this message to you.

Congratulations on your fine article and thank you in advance for your cooperation.

Yours sincerely,

Editorial Office

Evidence-Based Nursing

11.10.6 Faculty of 1000 Medicine

Dear G Parkes,

Your article (citation below) has been evaluated and rated by a respected peer within 'Faculty of 1000 Medicine'. We welcome your response to the evaluation, especially if you would like to comment on any criticisms made or if you feel

certain aspects of the article have been overlooked. Effect on smoking quit rate of telling patients their lung age: the Step2quit randomised controlled trial. BMJ, 2008 Mar 15.

Please click the following url link to be directed immediately to the published comments for free:

<http://www.f1000medicine.com/article/8kphldcjyfx71t2/id/1109230>

Feel free to involve your colleagues too and show them the feedback you have received for a piece of your work. This link will remain accessible when forwarded by email and your colleagues will also be able to view the commentary for free.

'The Faculty' is made up over 2000 of the world's leading researchers and clinicians. Their aim is to highlight and rate those articles that they think add important knowledge to the literature. Many of the world's leading medical institutions already subscribe. We look forward to your response to the evaluation, if you believe one would be of value to our readers. We advise that your response should be no more than 400 words. Please contact the other authors when preparing your response and email your responding comments to editorial@f1000medicine.com.

Please also note that, unless specified otherwise, it will be assumed that the response was sent on behalf of the entire author group. By sending your response or comment to editorial@f1000medicine.com you grant us the right to publish such response or comment. If you would like further access to our other content you can register for a free 3-week trial. Please see the link at the foot of this email.

We look forward to hearing from you.

With all best wishes,

Dr Pritpal S Tamber, MBChB

Managing Director, Faculty of 1000 Medicine

This study undertook a new direction for patients trying to quit smoking. The results demonstrated that supplying a patient with personal medical information

produced a surprisingly large positive effect, bigger than what might be expected from the usual nicotine replacement therapy trial. This RCT found that smokers given comprehensible 'lung age' information following spirometry were more likely to have quit smoking 12 months later than those given raw FEV1 (forced expiratory volume at one second) feedback (13.6% versus 6.4%, $p=0.005$, 95% confidence interval 2.2%-12.1%). These results need to be replicated before any firm conclusions are drawn. The study was underpowered to examine the extent of lung age deficit on quitting behaviour. This lack of power and the lack of a control arm (receiving non-personalised information about the effect of smoking on lung age) means it is impossible to dissociate the effect of receiving personalised risk information from the effect of simply receiving a verbal description of the effect of smoking on lung age. While interesting, further work is necessary before changes are made to clinical practice.

Robert West
with Jennifer Fidler
UCL, United Kingdom
PSYCHIATRY
Evaluated 19 May 2008

11.10.7 Wakefield District PCT

Subject: Lung age and screening for COPD

Date: Thu, 6 Nov 2008 16:49:59 +0000

From: Lisa.Chandler@wdpct.nhs.uk

To: Parkesko@hotmail.co.uk

Dear Dr Parkes,

I hope you don't mind me contacting you, I am sure you have had a lot of interest in the work you had published in the BMJ.

My background is as a Practice Nurse and Secondary Care Respiratory Nurse Specialist and I am now working as Respiratory Programme Manager in Public Health at WDPCT. We are attempting to implement a pilot with our local stop smoking service to identify patients with undiagnosed COPD. To encourage the stop smoking service to take part we are using screening spirometers, which

will also provide lung age, and quoting your research and increased quit success. We have been trying to get this work off the ground for the past 18 months and your work has opened a number of doors that had been closed to us previously, so thank you!! I am writing to ask if any patient or professional information was produced as part of your study and if so whether we would be able to negotiate the use of the information for the pilot we are undertaking. We are particularly interested in any patient information issued at the time of the test.

Thank you for your time. Regards
Lisa Chandler

Respiratory Programme Manager
Wakefield District PCT
West Yorkshire
WF10 5LT

11.11 Appendix 11. Presentation Slide



Telling smokers their lung age doubles quit rate

Dr Gary Parkes

Prof Trish Greenhalgh UCL

Joint UKFPCRO / PCRN Conference
29th November 2007

Slide 1 Introduction

The research question was:

Does

Spirometric lung age
testing
enable
people
to
Quit smoking?



Slide 2 Step 2 quit

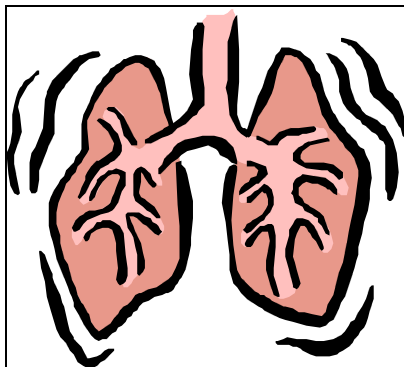
Key facts -East of England



- ⌘ Smoking Prevalence 23%
- ⌘ Over 200 deaths per 100 000 population due to smoking
- ⌘ 22 deaths per day
- ⌘ Total 2004-5.

Smoking in the East of England
March 2007: An update from the
Eastern Region Public Health Observatory
Total population 5.5 million people

Slide 3 Prevalence of smoking



Smoking causes
Lung Damage



- ⌘ 27% of smokers ¹
- ⌘ Aged over 35
- ⌘ have lung damage (COPD)

1. Van Schayck CP et al. BMJ 2002; 24(7350):1370 .



Slide 4 Lung damage

Biomarkers - Is Screening smokers worthwhile?



- ⌘ Cochrane review 2005 -
- ⌘ Due to limited evidence no definitive statement could be made about the effectiveness of biomarkers to promote successful smoking cessation¹

Reference

- (1) Bize R, Burnand B, Mueller Y, Cornuz J. Biomedical risk assessment as an aid for smoking cessation. [Review] [61 refs]. Cochrane Database of Systematic Reviews 2005;(4):CD004705.

Slide 5 Biomarkers Cochrane review 2005

- ⌘ Some recent evidence that detection of lung damage improves smoking cessation⁽²⁾

- (2) Bednarek M, et al. Smokers with airway obstruction are more likely to quit smoking. Thorax 2006; 61(10):869-873.

Slide 6 Bednarek- lung damage and smoking cessation

Quotes from NICE



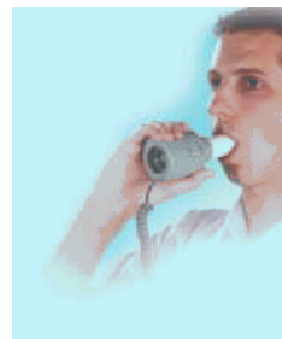
- ⌘ Smoking damage is detectable after 20 pack years of smoking - e.g.35.
- ⌘ the mean age of detection of COPD in the UK is 55
- ⌘ Use of spirometry can detect the presence of airflow obstruction earlier, even if no symptoms are present.
- ⌘ that the biggest factor that can have an impact on disease progression is smoking cessation .

Slide 7 Quotes from NICE

What is Spirometry?



- ⌘ blowing into a tube connected to a machine (spirometer)
- ⌘ Detects obstruction or restriction
- ⌘ Standard measurements include FEV1, FVC

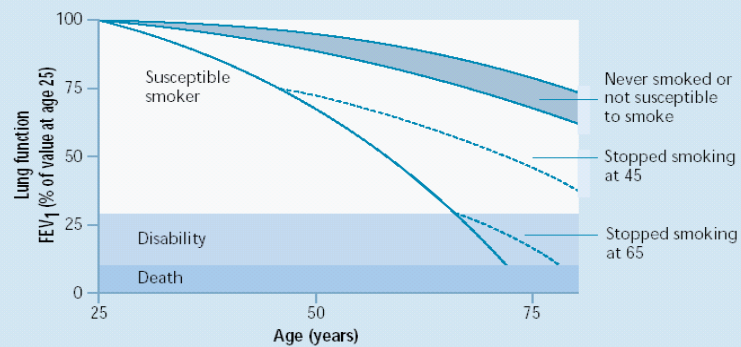


Slide 8 Spirometry

What is lung age?



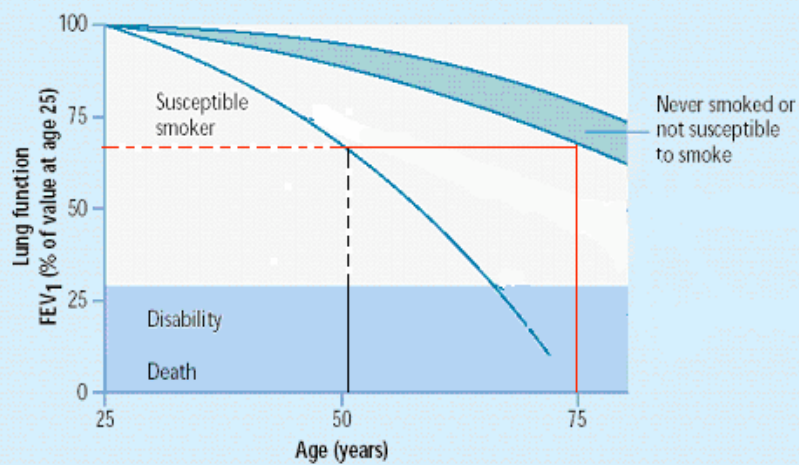
Smoking accelerates the normal age-related decline in lung function



In susceptible individuals, smoking accelerates the age-related decline in lung function, but this returns to the normal rate if the patient stops smoking (Adapted from Fletcher C & Peto R. *Br Med J* 1977; **1**: 1645-8.)

Slide 9 What is lung age?

What is lung age?



Slide 10 Illustration of lung ageing

Lung Age



- ⌘ A simple and clear way of explaining lung damage to patients.
- ⌘ FEV1 relates to aging of lungs. Smoking can give results equivalent to accelerated lung age

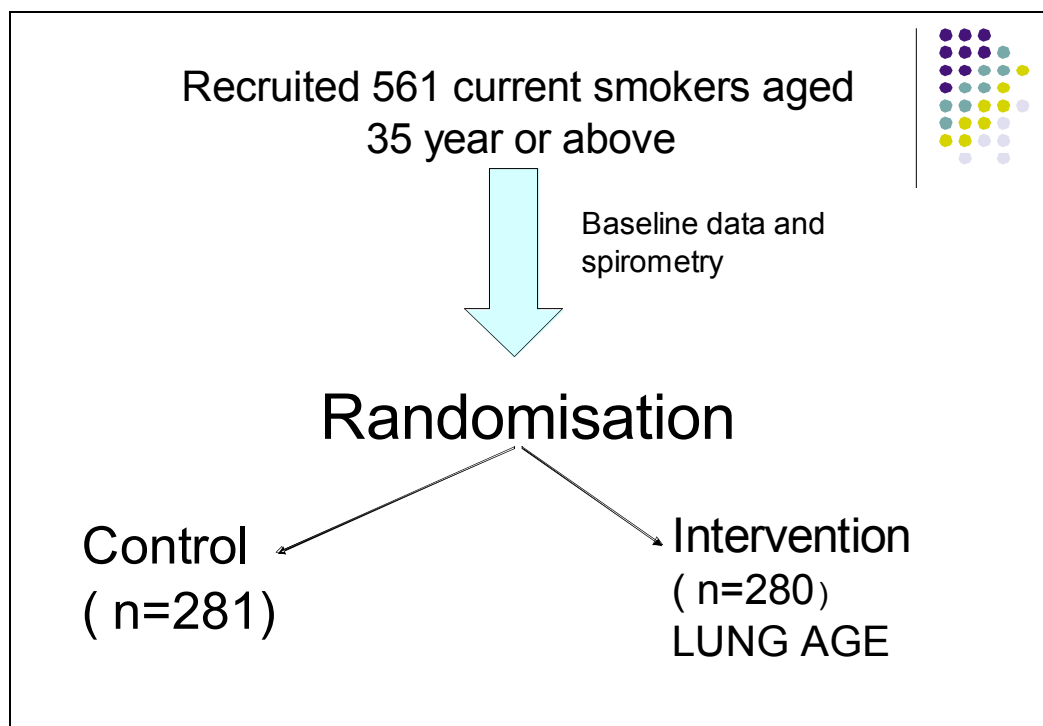
Slide 11 Lung age

Does Spirometric lung age Testing Enable Patients To Quit Smoking?



- ⌘ STEP 2 Quit Smoking study screened current smokers aged 35+
- ⌘ Randomised control study.
- ⌘ Results of lung function given to intervention group as 'lung age'.

Slide 12 RCT research question



Slide 13 Randomisation

Baseline characteristics <small>-(sample)</small>		
	Control	Intervention
⌘ Age	53	53
⌘ %Male	47	45
⌘ Pack years	30	31
⌘ Spirometry		
⌘ Abnormal	24%	27%
⌘ SGRQ	29	27
⌘ Cigarettes		
⌘ Per day	17	16

Slide 14 Baseline data

Intervention group



- ✧ Given result of test as LUNG AGE
- ✧ Shown graphic display
- ✧ Sent letter with results and advice on local smoking cessation resources

Slide 15 Intervention group

Control group



- ✧ Told that their measurements will be repeated after 12 months to see if their lung function has changed
- ✧ Sent letter with advice on local smoking cessation resources

Slide 16

12 months later



Control

follow up
249(88.6%)

Not followed up (n=32, 11.38%)

2 died
2 cancer/asbestosis
28 moved/failed to
respond

Intervention

follow up
249(88.9%)

Not followed up (n=31,11.07%)

1 died
1 cancer/asbestosis
29 moved/failed to respond

Slide 17 Control group

Results Quit rates



6.4% ^v 13.6%
control group ^v intervention group

(participants who failed follow up are assumed to be
smokers)

Slide 18 Quit rates



What causes the increased quitting?

- ⌘ Does more lung damage lead to improved smoking cessation?
- ⌘ $(\text{Lung Age} - \text{Age}) = \text{Lung age deficit (yrs)}$
 - ⌘ e.g. lung age 75 and real age 45
LAD= 30 years

Slide 19 What causes increased quitting



Lung age deficit (LAD) and quitting Intervention group

- ⌘ LAD= Spirometric lung age – chronological age
- ⌘ Quitters LAD= 8.0 years
- ⌘ Non-quitters LAD = 8.8 years
- ⌘ Quitting success is NOT related to severity of lung damage.

Slide 20 Lung age deficit

Other findings



- ⌘ 20% of the smokers had significant co-morbidity (CHD, DM, CVD, HT, Asthma)
- ⌘ A new diagnosis of COPD was made in 16% of participants

Slide 21 Co-morbidity

What this adds.



- ⌘ Screening and lung age estimation is useful for smokers of 35 years of age
- ⌘ NICE- guidelines for COPD and Smoking
 - ⌘ Update evidence
- ⌘ QuOF – new GMS contract could include incentives for screening all smokers

Slide 22 What this research adds

The end



Slide 23 The end

Bednarek M, et al 2006

- ⌘ Observational study
- ⌘ Non randomised
- ⌘ 4494 smokers from Poland
- ⌘ Those with lung damage shown a visual display.
- ⌘ Normal lungs - quit rate 12% v
- ⌘ Lung damage – quit rate 16.3%

(2) Bednarek M, et al. Smokers with airway obstruction are more likely to quit smoking. Thorax 2006; 61(10):869 -873.



Slide 24 Supplementary – Bednarek 2006

11.12 Appendix 12. -Masters dissertation.

**Does Spirometric lung age Testing Enable Patients to Quit Smoking?
(STEP2Quit Smoking)**

Gary Parkes

The Limes Surgery

8-14 Limes Court

Hoddesdon

Hertfordshire

EN11 8EP

Dissertation submitted in part-fulfillment of the Masters course in primary health care,

UCL, 15th August 2004.

Declaration - 'I, Gary Parkes, hereby declare that this dissertation is my own original work and that all source material used has been clearly identified and acknowledged. No part of this dissertation contains material previously submitted to the examiners of this or any other University, or any material previously submitted for any other examination.'

Signed:

Date:

Summary

Hypothesis tested in the pilot RCT

This pilot study tests the hypothesis that it is feasible to set up a study 1.using lung function screening of smokers in General Practice 2.using the concept of lung age as a sensitive biomarker of lung damage 3.using personal lung age as a communication tool to inform smokers about their health, in a way that may change rates of smoking cessation.

Hypothesis to be tested in the definitive RCT

Routine use of spirometry and lung age assessment in smokers improves success of smoking cessation in the setting of primary care.

Primary care clinicians are repeatedly encouraged in the medical literature to screen smokers for the detection of lung damage. Not all people are equally susceptible to lung damage from smoking and people with early damage may have no symptoms. The assumption is that smokers with damaged lungs can then be successfully targeted with smoking cessation programmes.

There is a lack of evidence that detection and knowledge of lung damage leads to an increase in smoking cessation. Therefore **The Aims** of the definitive study are to 'to strengthen the evidence base about the influence of personal

biomarkers on motivation to quit smoking' and to inform the debate about 'whether screening of smokers in primary care is worthwhile'.

Lots of research has identified medicines (patches, gum etc) that will help smokers to quit, who are ready for change, but the benefit of lung screening of smokers has so far not been researched in the NHS context.

The objectives of the definitive research study are to determine if :

1. Telling smokers their lung age changes motivation to quit.
2. Knowledge of lung age increases successful smoking cessation.

Lung function declines naturally with age. These changes can be detected and measured with a device called a spirometer. With reference to standard tables of normality for different ages (gender and height) the lungs of smokers may behave as if they are 'older' than normal. The thesis is that improving the knowledge about an individual's lung function and quantifying damage in an understandable way will improve success of smoking cessation.

This dissertation gives details of the background, literature review and a pilot project. The results and reflections on the processes involved are used to inform the development of a full scale research proposal which has led to a successful bid for a two year research grant for the definitive study.

I conclude that this dissertation demonstrates the feasibility and potential usefulness of a full research project as detailed.

This is the abstract of the Masters dissertation. If the full dissertation is required I can supply a copy on request.